The American Journal of Surgery

VOLUME 100 AUGUST 1960 NUMBER TWO

Presidential Address

The Sophomore Surgeon

WHEN you honored me by electing me to the Presidency of the Pacific Coast Surgical Association, I had experienced a long tenure as Recorder of our organization. In that office one of my responsibilities was to assist in the evaluation of most of the young surgeons on the Pacific Coast relative to their qualifications for membership in our group. For several decades I have concurrently discharged similar responsibilities for a number of surgical organizations covering Southern California or metropolitan Los Angeles. Most of the important training centers in the United States are represented in this group of men who are properly entering our ranks as qualified specialists in surgery. They embody the educational accomplishments of teachers of surgery like many among our members who consider the production of such highly qualified persons to be one of their most cherished responsibilities. My remarks will constitute an examination of some of the qualities of these recently trained surgeons. Furthermore, I hope to offer a few protestations for them relative to their residency experience and the professional milieu which is theirs on entering private practice. I have chosen to identify these new members of our profession as sophomores, using this term in a sequential sense. Also, I wish to draw upon the fundamental connotation of the term which is derived from two Greek words, the first, *sophos*, meaning wise, and the second, *moros*, foolish. I hope that this will not be objectionable if my accent is on the sapient implications of the sophical component. However, I intend some exploration of the terminal component as well.

Analysis of the fundamental qualities of our subject will reveal that he is the product of a program into which he entered as a selectee. It is therefore worthwhile to examine what he represents in terms of selectional criteria applied at the time of his appointment to a residency. What were the special qualities indicative of high potential as a surgeon which were found in him by those who made the appointment? I have spent much time examining candidates for residency. On each occasion it has been painfully apparent that the criteria available were exactly the same as those used by all other medical and surgical specialists. The appointees were those considered to be the most promising men, as measured by academic achievement in medical school and outstanding performance as interns. Therefore, the single determinant in their becoming surgeons was the fact that they, still quasi-lay persons in terms of surgical experience, had elected to apply for the specialty of general surgery. Their motivation and other personal qualities had been adequate to achieve the status of intern, but how completely were their abilities analyzable in terms of their adequacy for becoming surgical residents? Is it correct to assume that the ideal qualities of a surgeon had been identified beforehand by the examiners, and that they had learned to detect them in each person? If the candidate has demonstrated high academic achievement, the examiners can predict that such success will augur well for an adequate degree of mastery of the science of surgiology. Important as this will be, it in no way insures against maladroitness in the operating room. There has not been in the past, and there is not

now, a technic for measuring this latter quality in the candidate. Actually, the tendency is to underemphasize it in favor of the scholarly and scientific aspects of surgery. I do not belittle the latter, but am constrained to state that in most large private hospitals the avoidable serious surgical problems that arise are often dependent upon technical misadventure rather than upon lack of scientific knowledge relevant to the pertinent biochemical, physiologic and pathologic problems. The cardinal function of a surgeon is to operate upon patients. There is no substitute for technical excellence in surgiology. These foregoing considerations of such disparate non-academic factors as academic achievement and potential technical proficiency in the selection of surgical residents are submitted as examples to justify my complaint that in the mid-twentieth century we have no scientifically developed method for detecting those who are best qualified to become surgeons. Mindful of the vast investigative activities of the surgeons of the United States, is it not disturbing that we have been so unmindful of the science of our own professional procreation?

The attainment of an appointment moved our potential surgeon into his residency, and at least a symbol of his proximate goal was to become certified by the American Board of Surgery. Soon after starting his residency he may have wondered about some of the concepts underlying his training. While he had expected to become a surgical artisan, he basically considered himself to be entering into a scientific academic environment. More often than not this resident is in a university atmosphere. His perusal of the membership of the American Board of Surgery will have revealed to him that its members are all professors of surgery. At least five years later, if he passes their examinations, he will receive from them a certificate similar to that which he has already received from his State Board of Medical Examiners. Realization then comes that although he has been examined and passed by the select professors of the entire nation he is not to be rewarded by an academic mark of recognition. This arrangement, abetted by his own professors, is not made more acceptable when he considers the position he hopes to attain before starting private practice—that of one whose academic awards should distinguish him from the various guilds of surgical journeymen. It is my belief that the responsibility for correction of this situation lies with the educational institutions involved.

The foregoing contemplative considerations will shortly be brushed aside when, by fiscal necessity, the new resident will be forced to assimilate another part of his situation. Having acquired four years of college education, he has attended a professional school for an additional four years, and then served at least an additional year in postdoctoral training as an intern. This may well have been complemented by two years' experience in the medical corps of one of the armed services. This selected person, as a resident, will be continuously performing materially valuable services which fully utilize and require all his previous education. For this service he finds himself remunerated at a rate less than that of a plumber's apprentice in the same hospital. He cannot help but wonder why, in the past, the path of least resistance has been followed by his surgical fathers, and why the faculties that they constituted have not determined that society remunerate interns and residents with at least a living wage, even though this be far below the value of their services. Weak excuses rather than good reasons are given in explanation of this situation. Let us hope that, at least in the West, aurigerous reason may soon replace the acceptance of the fiscally asthenic traditions of the past.

When our sophomore surgeon enters the private practice of surgery, he will have been somewhat indoctrinated into the ethics of his profession. The anathema and the ethical proscriptions will have precluded itinerant and ghost surgery and involvement with commissions in any form. The first time the young man sees an older surgeon unnecessarily call in an internist to consult on a case, there will be sudden realization that fee splitting can take many forms. While abiding by the mores of respectable surgeons he will now meet some of the more subtle facts of life. To illustrate one, I will cite a personal experience. Early in my private practice a young internist friend called me and asked for the name of the best urologic surgeon in the city. He stated that a patient of his had a renal stone which required removal, and that, as the patient's physician, he wished to find for him the very best urologic surgeon available. As a young general surgeon, it was obvious to me that I would not have been consulted had the patient's problem been one

of gallstones. Therefore, the answer to this young internist involved asking him why, for the sake of consistency, he did not first refer his patient to the best local internist. The foregoing may seem somewhat trivial, but locked within it are several principles. Has our sophomore been taught what is morally right when he meets a problem that could be better handled by an older surgeon? Has he been led to believe, as some actually do, that having become a "Board Surgeon" he is completely qualified? This is a very convenient philosophy to accept, and for the acquisition of patients the term is very catching. More seriously, however, during residency the ethical concepts of the preceptor determine the young surgeon's answer to the foregoing question. These must necessarily have been reached in consonance with the basic truth that every surgeon must ultimately be replaced. The preceptor responsible for gradated training must, when a patient dies following a procedure performed by a resident, examine his own conscience with care. If in his own hands the patient probably would have lived, by what principle can he assuage his conscience? In difficult situations in private practice the implications for the young surgeon are the same except that now he makes the decision. Will he decide that what is right is that which is best for the patient? Intricately woven into the situation is the realization that if he matures through this particular experience, one life lost may have saved many in the future. While an answer to the foregoing may not be reasoned with complete lucidity in every situation, our sophomore will be, consciously or otherwise, reaching an answer more than once before he has been in practice a few years. His attitude should reflect that of his teachers, and should reveal his conviction that fiscal loss and pseudo-loss of face are secondary even though consequential. Our profession's answer to this problem involves an even heavier responsibility on the part of the senior surgeon in private practice who must, in spite of the abrasive effects of competition, be eager at all times to give counsel and help his young colleagues. This cooperation for the benefit of patients, with disregard of competitive relationships between older and younger surgeons, should be a canon of our profession.

Another ethical measure of the quality of our sophomore's training must be applied to his attitude toward experimentation. We shall assume that, as a part of his scientific education, he has properly developed a profound disrespect for empiricism, and that he has acquired familiarity with the principles of the scientific method. This has involved realization of the fact that science is a product of men's minds, and that the phenomena with which it deals are factual. The young surgeon has been trained to observe, to imagine and to reason. Also, he has learned that the product of reason must be validated by experimental proof, otherwise specious reason may go undetected when dealing with natural phenomena. Because the science of surgery is highly experimental, it is important that our young colleague appreciate the ethical principles of human experimentation as laid down by the Nuremburg Tribunal. The most fundamental of these states that full understanding and voluntary consent of the individual to be experimented upon are absolutely essential. Each initial application, by the originators, of many of our new contemporary surgical procedures approaches the status of an experiment, not always free of lethal potential. Most residents are exposed to this surgical progress at its source, and also are often associated with *bona fide* experimentation. When transposed to the status of a private practitioner, the young surgeon is bound by the rules of our courts. Actually this was true during his residency, but in his private practice he largely moves out of the investigative environment and, except for *bona fide* research, must recognize his new status. It is his duty to keep up with progress, but also he is bound by law to avoid casual experimentation. In addition, he must realize that although a practitioner may also be an experimentalist, the latter activity must be clearly identified as a separate endeavor conducted in accordance with the ethical rules for experimentation. The basic doctrine in his surgical catechism must be that of *primam non nocere*, meaning "first no harm."

Perhaps another facet of the recent resident's scientific educational background is worthy of examination. This involves the result of the program upon the methodology of his clinical investigation of the problems presented by his patients, and the logic with which he synthesizes the resultant evidence into a hypothesis that fits all the evidence. Of equally great importance is his manner in presenting his conclusions to the patient. Optimally this demands both a judicial mind and mien, particularly in

the sixty to forty decisions. This juridical combination of science and art should be considered a mandatory skill. Germane to these considerations it is justifiable to emphasize the profound need for the trainee's cognition of the intellectual sin committed in making an emotional decision. Experientially he may have witnessed such decisions in administrative ranks even at levels above his own departmental hierarchy. He may have also experienced this emotional problem in a very personal way when a patient of his own is beset by a serious postoperative complication which might be explained by an error in technic. If he is fortunate, he will recognize at the earliest possible moment in his career that emotion has no brains. When an emotional decision is abetted by wishful thinking, a tragic combination exists. A very realistic and somewhat pessimistic surgeon must endure more worry, but his judgements will be greatly superior to those of the optimist.

One other phase of the relationship between scientist and practitioner is worthy of mention. From the point of view of a scientist seeking to improve the cure rate of certain forms of cancer, it is appalling that the maximum cure rate is so tragically low. To a practitioner this is a clinical fact for which he is in no way responsible. Actually, the worse the cure rate the more important it is that he not err in diagnosis of the disease. The intelligent patient is morally responsible to find for himself the best medical help available to him. If he does this and his doctor makes a correct diagnosis and gives the best treatment known, both have fulfilled their responsibilities, regardless of the result. Today, as 1,000 years ago and for a long time to come, the result may be disastrous, but if it has been certified as being inevitable in the light of existing knowledge, the responsibility for the outcome is lifted from the shoulders of both doctor and patient. Therefore, in this sense, the lethality of an incurable disease is of no practical concern to a practitioner except as a clinical fact, however distressing it may be to him as a humanitarian.

The residency is an experience calculated to teach the selected person to function adequately as a surgeon. During this time it is not feasible to teach him how he will acquire a practice after completion of his residency. Therefore, the initial problem met after entering practice is one requiring a set of guiding principles. Actually, because the unit of practice is the patient and the patient's referring physician, the sophomore must establish the criteria by which such a discriminating unit judges him. It is by satisfying these criteria that the acquisition of patients is successfully achieved, a problem that at least in an overt way did not exist during residency because the teaching hospital acquired his patients for him. In satisfying each unit of practice that our sophomore acquires, and assuming his adequacy as a surgeon, the first essential quality so far as the referring physician is concerned is that of availability. If the surgeon is frequently hard to find, he is doomed to suffer seriously for this inadequacy. Not only must the surgeon be available but also he must at all times be physically and mentally fit, if he has accepted the rigors of giving continuous service. What does the newly acquired discriminating patient then properly expect, once he is seated in the surgeon's consultation room? He is there only because he has accepted or is about to accept the decision that surgery is the lesser of two evils, and that it should be performed only because it is deemed necessary. Implicit in the situation must be the patient's right of free choice of surgeon. Furthermore, the patient wants the problem solved with a minimum loss of time, and the operation performed for a reasonable fee and without unnecessary hospital charges. In addition to these material factors, the patient expects to be cared for with gentleness, sympathy, precision and frankness, and in such a way that he suffers a minimum of pain and discomfort. Emotionally the entire situation is basically distasteful to him. These factors should be fully recognized by the surgeon. Fiscal matters should be discussed. Initially the patient should know within what range a fair fee will fall. The young surgeon may well reflect his training in the matter of the patient's hospital costs. This training should have emphasized that every order he wrote on a patient's chart constituted the filling out of a blank check, given to him in trust. In the category of wasting time it is worthy of comment that in today's medical school curricula great emphasis is being placed by the behaviorists on total knowledge of the patient and his environment. The acquisition of this knowledge may be inordinately expensive in terms of time. Although total study of patients is clearly necessary in some situations, the wise surgeon will learn to eliminate all that wastes

time, to the benefit of both the patient and himself. Time is the only real capital possessed by both the doctor and patient. If the surgeon sees twelve patients twice daily in the hospital and wastes only five minutes during each visit, he has squandered two hours. These two hours applied in daily study would, in a period of years, determine that he would have risen to and maintained the status of a scholarly practitioner. For the young surgeon I wish to quote Stephen Vincent Benet who wrote, "Life is not lost in dying; life is lost minute by minute, day by dragging day, in all the small uncaring ways . . . "

A few more considerations should be mentioned in outlining as I have some of the desirable qualities with which the young surgeon may well be indoctrinated. Another is his willingness to tithe to his profession by giving freely of his time, as repayment to society, for the upbuilding of that profession. He should by this time have been made to realize that society has actually, through its schools as well as throughout his surgical learning experience, contributed heavily to his education. In fiscal terms it has contributed the major portion of this education. Throughout his earlier years he may frequently have asked, "Why don't they do this and why don't they do that?" When he is older, the answer is, "You are now they." He then realizes fully that surgery is responsible for itself, and that this capability is dependent upon the voluntary dynamic assistance that every mature surgeon must willingly give. If our new practitioner is sufficiently observing in these matters he will be awakened to some of the delicacies of the interlocking relation between self-interest and altruism, and recognize that overt altruism is a cloak for self-interest. Ultimately, examination of his own conscience will reveal that these qualities are not ambivalent, and that the true test is the degree of sincerity that exists in the altruism. If proved wholesome by this test our young surgeon's idols will not be found to have feet of clay, and he will be stimulated to take his place in the ranks of those striving to serve their profession.

Lastly, and perhaps most important, is the need for full realization by our sophomore that in terms of years of study, most of his surgical education still lies ahead and that henceforth he must be his own teacher. There will be no professional bloom until he has so matured that he has learned to love learning for learning's sake. Attainment of this goal will secure his position as a scholar and as a respected member of a learned profession rather than that of a craftsman whose academic interests have long since disintegrated. The intellectual gifts with which a man is born reflect no achievement on his part; it is his augmentation of them which may be to his credit.

I have attempted to present a review of certain matters relevant to the educational experience of young surgeons, with the belief that such a discussion of them will bear fruit. No doubt it has been apparent that many of the problems about which I have spoken involve errors of which I myself have been guilty in the past, and that my bibliography has been mostly one of personal references. May this admission clear me of any semblance of hypocrisy. As retiring President it is my hope that this year has moved the Pacific Coast Surgical Association importantly upward in its maturation, in stride with our great nascent Western empire.

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The Syndrome of Mineralocorticoid Excess: Primary Aldosteronism

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INTEREST in endocrinology in general and in the adrenals in particular increased after World War II under the stimulus of improved physicochemical and biologic methods for the identification of hormones. Many hormones have been isolated from the adrenal cortex during the past fifteen years, but four hormones originating in the human adrenal cortex account for almost all of its recognized biologic effects. These hormones are hydrocortisone, secreted daily in 10 to 20 mg. amounts; aldosterone, secreted daily in amounts of 0.2 to 0.4 mg.; small amounts of corticosterone, and small amounts of C_{11}-oxygenated 17-ketosteroids. These hormones can now be found regularly in the adrenal cortex, adrenal venous blood, adrenal incubates, and their degradation products can, under appropriate technics, be recovered from the urine. These hormones tend to produce rather specific physiological changes, and today hydrocortisone is referred to as a *glucocorticoid* for it affects primarily carbohydrate, fat and protein metabolism; aldosterone is termed a *mineralocorticoid*, for it affects essentially sodium, potassium and water metabolism; the remaining adrenal cortical hormones affect primarily androgenic and estrogenic functions. Even the histologic origins in the adrenal cortex are being defined, and the zona glomerulosa produces essentially aldosterone, the zona fasciculata the glucocorticoids, and the zona reticularis the androgenic and estrogenic hormones.

The clarification of functioning or hormonal disorders of the adrenal cortex in man is resulting from our increased knowledge of basic biochemistry and physiology. Clinical disorders involving obesity, hypertension, hirsutism, menstrual disturbances, hypokalemic alkalosis, polyuria, glycosuria, osteoporosis and infertility were first described as Cushing's syndrome, and were believed to be caused by a pituitary tumor. Adrenal cortical tumors and bilateral adrenal hyperplasia were discovered in some of these patients in the late 1930's. The interrelationships of the adrenal and pituitary glands, the function of ACTH and adrenal cortical hormones, and identification of urinary metabolites of the 17-ketogenic and 17-ketosteroids helped clarify the adrenal hormonal disturbances during the 1940's, but extensive research during the past decade has furthered the identification of adrenal corticoids into the three main categories: the glucocorticoids, the mineralocorticoids, and the androgenic and estrogenic corticoids.

The identification of aldosterone as a separate adrenal cortical hormone followed the brilliant investigations of Luetscher [1] and Simpson [2]. In 1950 Deming and Luetscher [1] reported the bioassay of a material like desoxycorticosterone in the urine, desoxycorticosterone being a synthetic steroid affecting salt and water excretion by the kidney. Simpson et al. [2] in 1952 discovered the secretion of a salt-retaining hormone by the mammalian adrenal cortex. The isolation of this hormone followed improvements in chemical

technics, for the initial chemical methods resulted in destruction of the hormone. Using partition chromatography, which permitted separation of the hormone without excessive loss or damage, a group of experts [3] combined their efforts to identify the unique structure of this substance. It has been called aldosterone.

The measurement of aldosterone has been and remains difficult. Either a biologic technic can be employed in skilled hands, or a physicochemical technic can be developed in refined chromatographic laboratories [4]. Despite difficulties and inaccuracies of analysis, the excretion of aldosterone has been demonstrated to be increased in states of sodium depletion, nephritic syndrome, congestive heart failure, liver disease with ascites, idiopathic edema, and occasionally in hypertension, stress or severe edema. It is decreased in hypopituitarism and Addison's disease. It remained for Conn [5], however, to find and report the first case of primary aldosteronism. In 1955 he described the syndrome of hypertension, hypokalemia and metabolic alkalosis associated with excessive production of the mineral-regulating steroid of the adrenal, aldosterone. Lieberman [6] has reported the secretion of corticosteroids other than aldosterone in Conn's syndrome, and it is probably best to describe the syndrome as one of mineralocorticoid excess.

The purpose of this paper is to review for surgeons the pertinent aspects of the syndrome of mineralocorticoid excess. The cases discussed were kindly referred through Dr. Luetscher's group, and the detailed and scholarly presentation of these cases has been made elsewhere. Only pertinent clinical features will be presented.

BASIC PHYSIOLOGY OF ALDOSTERONE

The primary site of action of aldosterone is on the renal mechanisms for absorption, exchange and excretion of salt, potassium and water. Sodium is preferentially retained by the tubule, and potassium ion is lost. The body water is altered indirectly, for the urine remains dilute. Hydrogen ion is also exchanged for some sodium in the distal tubule, so that even though large amounts of potassium ion are lost there is also an increased output of hydrogen ion in the urine.

The regulation of aldosterone secretion is less influenced by the pituitary than glucocorticoid secretion. Depletion of body sodium is the prime stimulus to aldosterone secretion. ACTH and the other corticoids tend to stimulate mildly the secretion of aldosterone. Water deficit, decrease of the circulating extracellular fluid, and loss of blood increase aldosterone secretion. An excess of potassium or "potassium loading" also augments aldosterone secretion.

On the contrary, aldosterone secretion is decreased by a body excess of sodium, an excess of water, increased blood volume and potassium depletion.

The effects of aldosterone have been more clearly elucidated by the study of aldosterone administration to both normal and edematous patients. The normal patient responds to the administration of aldosterone by an initial retention of sodium, followed by an escape with a return to a normal sodium balance. Urinary potassium increases, and the diastolic blood pressure rises. The edematous patient, according to Nelson and August [7], responds by an initially greater sodium retention, but no escape follows. The loss of urinary potassium does not increase significantly, and the blood pressure changes are variable. Therefore, there are probably two sites of action of aldosterone on the tubules of the kidney, related to the existing concentration of sodium in the tubule. The absence of sodium in the distal tubule in the edematous patient prevents any exchange with either potassium or hydrogen ion, thereby preventing loss of urinary potassium. Also in the edematous patient the excess sodium reabsorption occurs proximal to the site of potassium exchange.

These important relationships of the concentration of sodium ion in the tubule with respect to loss of potassium ion and hydrogen ion in aldosterone excess explain the importance of altering the sodium and potassium contents of the diet and studying clinical cases of aldosteronism.

In summary, aldosterone leads to an excessive loss of potassium and hydrogen ion from the distal tubule, probably exchanging them for sodium. Sodium retention occurs. The urinary ammonia excretion is increased, and the urine remains alkaline or neutral despite the increased loss of hydrogen ion. The urine remains dilute, even with the administration of Pitressin.® In time this may lead to a relative hypernatremia with a mildly decreased blood volume. Renal damage ultimately occurs with retention of nitrogen and failure of sodium conservation.

Aldosterone secretion is depressed by the body depletion of potassium and the excess of sodium.

The measurement of aldosterone in the urine is extremely difficult, requiring special bioassay technics or elaborate chromatographic separation. The value must also be interpreted in the light of the state of potassium depletion or sodium excess, both of which decrease its output.

ALDOSTERONE ANTAGONISM

Recently a series of new synthetic steroids has been produced which are reported to be antagonistic to the renal effects of aldosterone in adrenalectomized rats and dogs. Chemically they are characterized by having a spirolactone group on the seventeenth carbon of corticoid ring. Studies in man with SC 8109 [8] have shown that this aldosterone antagonist does block the effects of aldosterone not only on sodium and potassium excretion but also on urinary ammonia and titratable acidity. Normal subjects whose aldosterone output has been suppressed by a high sodium diet will not respond, but, on a low sodium diet with high aldosterone output, SC 8109 increases urinary sodium excretion. The drugs apparently antagnoize the peripheral effects of aldosterone, and can be expected to act in patients with excessive aldosterone production. The drugs have inhibited the action of aldosterone in primary aldosteronism, promoting sodium loss and some retention of potassium. They have also reversed aldosterone effects in congestive heart failure, cirrhosis with ascites, severe edema and the nephritic syndrome. Their use in the latter states is more difficult and questionable than is their use as a diagnostic adjunct in primary aldosteronism.

DIETARY VARIATIONS AND ALDOSTERONE

Effect of a Low Sodium Diet: On a very low intake of sodium, in the vicinity of 10 to 15 mEq. per day, little or no sodium reaches the distal renal tubule, and exchange with potassium does not occur. Potassium retention is favored by a diet very low in sodium.

If the kidneys are essentially normal, no sodium will be lost in the urine on the low sodium diet. This distinguishes primary aldosteronism from "salt-losing" nephritis or secondary aldosteronism, for urinary sodium loss continues in "salt-losing" nephritis.

Even in the presence of moderate kidney damage in primary aldosteronism, body repletion of potassium occurs on a very low sodium diet. This influences the kidney favorably, and the kidney does not excrete any sodium on the low sodium diet.

Effect of a High Potassium Diet. Potassium loading leads to retention of potassium by the body. If 150 mEq. per day of potassium are administered, over 100 mEq. per day will be retained for several days, despite a continued fairly high loss of potassium in the urine. Aldosterone excretion also increases under potassium loading, and this may be detected by urinary assay.

SYNDROME OF MINERALOCORTICOID EXCESS

Excessive mineralocorticoid action by the adrenal cortex arises primarily from excessive aldosterone output. Other adrenocortical hormones may also influence mineralocorticoid action, and it is preferable to view the clinical syndrome as mineralocorticoid excess rather than as pure primary aldosteronism. Furthermore, studies of the hormone production by the tumors we have removed under the direction of Dr. Luetscher have demonstrated hormones other than aldosterone to be elaborated in adrenal incubates [9]. Hence, the clinical picture will be described as mineralocorticoid excess.

The chief complaints of patients with mineralocorticoid excess are headaches, thirst and polyuria. Lassitude and weakness are common. Paralysis or periodic paralysis occurs less commonly, expecially in the diet rich in sodium. Tremors and muscular spasms occur.

Physical examination discloses a hypertension with attendant secondary complications. Trousseau's and Chvostek's signs may be present.

The characteristic laboratory finding is a low blood potassium. The bicarbonate may be elevated, and the serum sodium is variably elevated.

The association of hypertension and hypokalemia should invariably suggest the diagnosis. Every hypertensive patient must have a serum potassium study if these cases are to be discovered and cured. The association of hypertension and hypokalemia demands further investigation for the syndrome of mineralocorticoid excess.

The urine is dilute, and concentration does not occur even when Pitressin is administered.

The urinary ammonia is increased, but the titratable acidity is low, and the pH close to neutral or alkaline.

Further studies include observation of the sweat and salivary sodium and potassium. The sweat and salivary sodium is low, the potassium high, and the sodium ratio is low. If tissue analysis should be done, the tissue sodium would be high and the potassium low, for there is a general body excess of sodium with potassium depletion.

Ultimately renal damage occurs with vacuole formation and tubular degeneration. Casts appear in the urine, and calcium deposits are found about the tubules.

SPECIAL STUDIES IN ALDOSTERONISM

The measurement of excessive amounts of aldosterone in the urine would confirm the diagnosis. However, this is generally difficult or not available. Further substantiation of the diagnosis is gained through the following special studies.

Potassium Repletion. A diet of 150 mEq. of potassium every twenty-four hours is given. The body retains over 100 mEq. every twenty-four hours for a few days despite the continued high renal loss of potassium. This indicates a total body deficit of potassium, confirmatory evidence of mineralocorticoid excess.

Low Sodium Diet. A diet of 10 mEq. of sodium is given every twenty-four hours. Potassium is retained, for little or no sodium reaches the distal tubule for exchange. If the kidneys are normal, no sodium appears in the urine, thereby excluding the diagnosis of "salt-losing" nephritis or secondary aldosteronism. If the kidneys are moderately or mildly damaged, the attendant potassium repletion often improves kidney function so that minimal or no sodium is lost in the urine.

Urine Studies. The specific gravity of the urine remains low, around 1.010, despite the administration of Pitressin. The primary ammonia is high; the titratable acidity is low, and the pH remains neutral or slightly alkaline, despite increased excretion of hydrogen ion.

Sweat and Salivary Studies. The sodium in sweat and saliva is low, the potassium high. The ratio of sodium to potassium is low.

Aldosterone Antagonists. Administration of amphenone and Spironolactone,® aldosterone antagonists, increases the urinary sodium excretion and diminishes the urinary potassium loss. Body repletion of potassium tends to occur with clinical improvement.

Retroperitoneal Nitrous Oxide. An adrenal tumor may be demonstrated by this technic, but in practice retroperitoneal gas studies have not proved too helpful. We have not advised them in our cases.

The study of renal function and potassium balance by these special methods has proved as reliable in diagnosis as the measurements of aldosterone. These studies are simple, readily available and diagnostic.

DIFFERENTIAL DIAGNOSIS

The association of hypertension and hypokalemia is almost pathognomonic. The differential diagnosis includes a few other causes of hypokalemia, most of which can be readily distinguished by the attendant clinical and laboratory studies.

Hypokalemia can result from inadequate intake of potassium, faulty intestinal absorption, or abnormal intestinal losses through intestinal fistula and diarrhea. Chronic overuse of laxatives can produce hypokalemia and metabolic alkalosis. The history and physical findings generally make these causes of hypokalemia obvious.

Abnormal potassium loss may occur in the urine of patients taking diuretics, as chlorothiazide. The association of hypertension, hypokalemia and metabolic alkalosis could occur, but the history should prevent this error in diagnosis. Aldosterone excretion, if measured in this situation, would be low.

Other causes of urinary potassium loss, such as excessive cellular breakdown, diabetes mellitus, acidosis and dehydration, are apparent on routine physical examination.

Renal tubular acidosis, with its dehydration and renal sodium loss, may mimic aldosteronism, but it should be ruled out by the associated hyperchloremia, acidosis, hypophosphatemia, and tendency for serum sodium and calcium to be reduced.

"Salt-losing" nephritis or secondary aldosteronism is distinguished by the low sodium diet. These patients continue to excrete sodium in the urine on the low sodium diet.

SURGICAL CONSIDERATIONS

Preoperative. Potassium repletion by diet prior to surgery seems desirable. Zimmerman et al. [4] report an operative fatality in their

first operated cases of primary aldosteronism which they attribute to failure of potassium repletion prior to surgical intervention. Potassium repletion further increases the aldosterone excretion, but a potassium intake of 150 to 300 mEq. every twenty-four hours invariably results in total body repletion. The state of potassium repletion can be judged by balance studies, serum potassium levels or measurements of twenty-four–hour urine excretions. The CO_2 combining power and serum potassium should preferably be normal prior to operation.

Operative. The transabdominal approach is preferred so that both adrenals may be inspected simultaneously. A transverse upper abdominal incision gives adequate exposure. The entire adrenal on both sides must be mobilized (we almost missed a tumor located behind the left adrenal gland). This tumor could not readily be palpated prior to complete adrenal mobilization. The disorder generally results from a solitary tumor. If a solitary tumor is found, the tumor alone should be removed.

If an adrenal tumor is not found, the preferable treatment at present would seem to be total adrenalectomy on the right side, followed by removal of 90 per cent of the left adrenal at the initial operation. The secondary operations on the left adrenal are simpler than on the right. Thus far, several cases of mineralocorticoid excess due to hyperplasia have been reported [10], and cure has resulted from complete removal of one adrenal with extensive subtotal resection of the other. No postoperative corticoid therapy has been necessary in these patients after the initial postoperative period.

Zimmerman [4] warns against the use of curare or drugs like curare during the operation because of the potentiating effects of potassium deficiency on compounds like curare. However, we have employed general anesthesia with these compounds without difficulty.

If the tumor is large and difficulty in exposure is anticipated or encountered, the use of a thoracoabdominal approach is recommended. A very difficult operation on a large adrenal tumor can be rendered very simple by the thoracoabdominal approach.

We have not employed any cortisone preoperatively or during the operation in these cases, but it should be available as the hydrocortisone for intravenous use in the event of any unexpected hypotension, fever or shock during the operative procedure. If desired, 100 mg. of cortisone can be given the night before and the morning of surgery to allay apprehension about insufficiency of the other cortical hormones.

Postoperative. No difficulty has been encountered in the regulation of fluids and electrolytes or in the control of shock, fever or anemia. Hydrocortisone and nor-epinephrine should be available for intravenous use in the event of unexplained shock or fever, signs of adrenal cortical exhaustion. The postoperative fluids and electrolytes have required no special attention, and a normal diet can be resumed within a few days.

SUMMARY

Three patients with mineralocorticoid excess have been diagnosed, operated upon, had an adrenal cortical tumor removed, and been cured of primary aldosteronism. The clinical symptoms have been headache, thirst, polyuria and weakness, lassitude and periodic paralysis. The clinical signs have been hypertension and occasionally signs of muscular irritability and weakness, such as a positive Chvostek and Trousseau sign.

The association of hypertension and hypokalemia strongly suggests the diagnosis of mineralocorticoid excess. Confirming evidence is found in the dilute urine with increased ammonia excretion, low titratable acidity and neutral pH. The sweat and salivary sodium content is low, the potassium high. Body potassium deficiency, as demonstrated by potassium loading, and the renal response to a low sodium diet, namely, no loss of sodium in the urine and body potassium repletion, are reliable diagnostic tests, and far simpler than the measurement of urinary aldosterone at present.

Preoperative management includes potassium repletion by a high potassium diet. A transabdominal operative approach is advised. If a tumor is found, it is removed; if hyperplasia is found, total adrenalectomy is performed on the right side and subtotal adrenalectomy on the left. All patients have been cured by operation, and all had isolated adenomas.

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DISCUSSION

Clarence Stafford (Los Angeles, Calif.): Dr. Richards has emphasized both the basic physiology and the clinical features of this rather complicated syndrome.

In reviewing six cases that came through our medical school I tried to reduce the clinical features to a least common denominator. All the patients in this series had both headache and hypertension. Headache was associated in some instances with other symptoms, such as nausea and vomiting, and in one case the presenting symptom was "blurring of the right eye." One sees how difficult it is to make a diagnosis with a type of symptom like this.

In most of the cases, weakness seemed to come in intermittent attacks. None of these patients had actual paralysis, but they did have weakness, noted mostly in the extremities. In no case was there evidence of tetany.

Nocturia was present in all except one patient. It varied anywhere from two or three times a night to five or six times, and the urine, as previously mentioned, had a low concentration.

Increased thirst was not as marked a symptom as one might expect. The patients did not complain of it so much before the operation, but after surgery, when they were relieved of this symptom, it was more noticeable in retrospect.

The hypertension varied from 190 to 230 mm. Hg. In reviewing other cases of aldosteronism which have been reported, it is apparent that any one or more of these symptoms can be absent, but in this particular group headache and hypertension were present in each patient.

Some commonly used laboratory procedures which are available in any hospital form a valuable triad for screening suspected cases. These tests are serum potassium, serum sodium and CO_2 combining power. If we are to diagnose these cases, we must have some sort of screening mechanism for all patients with hypertension and headache. If this group represents the accurate range of age, the screening should apply especially to those who are young and middle-aged. All these patients except one girl (sixteen years of age) were in their thirties or forties.

Serum potassium was definitely decreased. The values were usually between 2 and 3 mEq., whereas the serum sodium and CO_2 combining power were either slightly elevated or close to normal, at least not nearly as marked as the constant decrease in serum potassium.

If a patient has headache and hypertension and there is a marked decrease in the serum potassium, other tests should be made. It is hardly practical to use the aldosterone level as a screening test on everyone with hypertension. This is rather expensive and quite complicated, and can be reserved for those in whom the rest of the tests seem to point toward this condition. The ratio of sodium to potassium in the saliva also seems to be a very reliable confirmatory test.

Surgery revealed that each patient had a benign adenoma. They all recovered, and the symptoms subsided except in one patient who had hypertension for over thirty years. In this case the hypertension was decreased only a slight amount. The others were completely relieved of their symptoms.

I would like to emphasize, in conclusion, that if we can influence the internists to screen their patients with hypertension by simple laboratory procedures, and then move into the more complicated confirmatory procedures in suspected cases, we will be able to find more of these very interesting cases.

Arthur J. Hunnicutt (Oakland, Calif.): These cases are still so comparatively few in number that we are not justified in speaking of percentages. I would like to comment on a few patients with this "sodium retention syndrome" who have had some unusual signs and symptoms.

One patient was operated upon very recently. An aldosterone tumor about 1.5 cm. in diameter was found in the left adrenal, as were three other microscopic tumors of the zona glomerulosa. It makes one wonder about the fate of the other adrenal even though it was grossly normal.

Three months before this operation, the patient had given birth to a baby. Her blood pressure was normal during pregnancy and delivery. Twelve

days after delivery her blood pressure went up to about 160/118 mm. Hg. Weakness, tetanic contractions of the muscles and severe headaches developed, and her weight increased.

Aldactone® was given to her for three days under the direction of Dr. Harry Balch. The serum potassium returned to normal; the serum sodium remained approximately the same. She lost less of her urinary sodium. Her blood pressure, which had been up to 160 mm. Hg, returned to nearly normal (130/90 mm. Hg). Her weight decreased about 20 pounds.

In 1957, after the birth of a baby, she had a transitory episode (about two weeks) of headaches, gain in weight, lethargy and weakness. This was controlled with diuretics. From then until after the birth of this last baby she was essentially well.

It is said that these people have edema but they do not have pitting edema. This patient had pitting edema.

Two patients had all the clinical and laboratory findings of aldosteronism except increase in blood pressure. In each of these, the pathologist described "a localized area of peculiar staining cells" in the superficial layer of the adrenal.

In both these patients the symptoms regressed—not entirely but quantitatively—to the point where they could eat a reasonably well rounded diet including a reasonable amount of sodium without getting edematous, and their lethargy and weakness regressed.

The fourth case was a sixteen year old girl with rather marked sodium retention. As her weight would increase she would get severe epileptic convulsions. She had had about three menstrual periods during her life.

On exploration her adrenals were grossly normal. She had classic so-called Stein-Leventhal or polycystic ovaries. A large wedge resection was performed on each ovary. Following this, her weight regressed; she is not on any sodium restriction, is having normal menstrual periods, and her epileptic attacks are now few and minor.

I think the last case illustrates the so-called functional or stress cause of sodium retention. This young lady was examined surgically and we found nothing abnormal in the ovaries or adrenals. Her symptoms continued. One year later she had a hysterectomy. The ovaries were not removed. Her doctor wrote me that following this surgery, all her signs and symptoms of sodium retention disappeared and she was perfectly healthy.

One of the very real problems in patients with sodium retention is trying to determine which should undergo surgery and which should not.

Maurice Sokolow (San Francisco, Calif.): As the only internist in the group, I would like to call attention to one other source of difficulty in recognizing this syndrome, which actually has been the greatest source of trouble in our hypertension clinic. This concerns patients who have been taking chlorothiazide. It is very uncommon for patients with hypertension not to be given chlorothiazide by their local physician. In patients who have had this drug for a period of time, the serum potassium frequently falls. The patients complain of weakness, the serum sodium may be essentially normal, there may be a hypochloremic alkalosis and the CO_2 may be elevated. Therefore, the patient may superficially resemble in all respects a patient with aldosteronism. Before the diagnosis is established, the drug should be stopped for a period of at least a week or two to allow repletion of potassium to occur.

In some cases, potassium depletion occurs out of proportion to sodium depletion, and the electrolyte disturbances may be confusing.

Dr. Richards, I am sure, has run into this problem, but I think it is important to call it to your attention, as he did not mention it.

Victor Richards (closing): Dr. Sokolow has emphasized the salt-losing effects of diuretics on aldosterone production. Similarly, excessive production of aldosterone occurs after prolonged administration of moderate laxatives. These causes of secondary aldosteronism must be distinguished from primary aldosteronism, and, of course, should not be subjected to surgical intervention.

Surgeons must be interested in hypertension, for more causes of hypertension which are amenable to surgical cure are being found. So far as this particular disease is concerned, the clue to diagnosis is the association of hypertension plus hypokalemia. All hypertensive patients should have their serum potassium measured. Hypertension combined with hypokalemia should arouse the suspicion of the diagnosis of primary aldosteronism. It is a curable type of hypertension.

Arterial Reconstruction by Endarterectomy

FOUR-YEAR FOLLOW-UP STUDY

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THERE has been wide acceptance of the principles of arterial reconstruction, but there is no firm opinion as to the form that reconstruction should take. There is, in other words, some disagreement as to the relative merits of thromboendarterectomy and of bypassing with fabric prostheses.

Some factors that must be considered in order to come to a proper decision as to the choice of procedure must include: (1) the technical ability of the particular surgeon to perform the procedure on the particular patient, (2) general status and prognosis of the patient, (3) immediate results of the operation, and (4) late results of the operation.

Technical matters have been discussed extensively elsewhere by us and by other authors. It is with the second and fourth points that the following paper will be concerned.

This paper cannot properly present a controlled series comparing thromboendarterectomies and bypass prostheses, for bypassing technics have been used only infrequently by us for reconstruction of obstructed arteries; bypassing has been used only in selected cases in which a poor prognosis existed from the point of view of both the general and local circulation, and therefore cannot fairly be compared with the more favorable cases in which endarterectomy was chosen. We are not aware of any large series in which the two technics have been applied at random to achieve a controlled study. We can offer, then, no more than a résumé of the long-term results with thromboendarterectomy in our hands.

Recognizing that the comparisons may not be truly accurate because of the differences in original material of each author, we, nevertheless, hope to offer this group for later comparison with results from other clinics in which prosthetic technics were employed.

The present series consists of forty-four patients operated upon by us and the staff members of the Wadsworth Veterans Hospital and University of California Hospital, using the technics described by us [1–3] between October 1951 and the end of 1955. This excludes several operations performed prior to this date in which fallacious approaches were used with poor results. The technics described are almost identical with those described by Wylie et al. in 1951 [6]. At least four years have elapsed since the last patient in this series was operated upon. Not all patients could be followed up for the full period.

There were six initial failures among the forty-four patients; thirty-eight patients were, therefore, available for protracted follow-up study. This initial failure rate is much higher than should be tolerated at the present.

Ten patients were lost to follow-up study within three years; three of these patients died, one at six weeks from pulmonary embolus, and two at one and two years, respectively, from coronary occlusions. In these three cases a satisfactory local result existed at the time of the patient's death. There were no known failures in the other seven cases. (Table I.)

Five patients were followed up from three to four years. (Table II.) Three of these patients died. One patient with an aortic-iliac obstruction died of sarcoma after a satisfactory arterial result. Two patients died of coronary artery disease (one of whom also had a renal infarct) after satisfactory femoral and femoropopliteal reconstructions.

Two patients were known to be living when lost to follow-up study. One of these patients

TABLE I
SURVEY OF FORTY-FOUR PATIENTS OPERATED UPON BETWEEN 1951 AND 1955

Initial failures	6
Lost to follow-up study in first three years (no known failures)	7
Dead in first three years	3
Available for follow-up study after three years.	28

underwent femoral reconstruction which failed gradually after two years, and the other patient underwent aortic-iliac-femoral reconstruction which failed in its distal portion to some degree but remained good enough for the patient to play golf. Distal pulses disappeared but those in the central portion of the reconstruction remained.

Thirteen patients could be followed up between four and five years. (Table III.) Seven of these patients were originally treated for pure aortic-iliac occlusions. Six of the seven maintained patency of the endarterectomized segment and one later had not only a separate femoral occlusion but also a cerebrovascular accident. In the one failure the patient had in retrospect an inadequate attack on a localized segment of complete occlusion in an aortic and iliac tree plagued with multiple stenoses which were not removed.

Three patients with pure femoral disease maintained their femoral reconstructions; one, however, died of hypertensive vascular disease, one was never relieved completely of his leg complaints although good pulses were restored and maintained, and one was clinically unchanged despite late occlusion of the tree distal

TABLE III
FOUR- TO FIVE-YEAR FOLLOW-UP STUDY OF THIRTEEN PATIENTS

Site	Local Result	General Result
Seven aortic-iliac lesions	6 Good	One cerebral and femoral occlusion
	1 Failure	Associated cardiac and separate femoral disease
Three femoral lesions	3 Good	Death from hypertension
		Distal occlusion
		Good
Three aortic-iliac-femoral lesions	3 Good	Two femoropopliteal occlusions

TABLE II
THREE- TO FOUR-YEAR FOLLOW-UP STUDY OF FIVE PATIENTS

Arterial Site	Local Result	General Result
Aortic-iliac	Good	Death (Sarcoma)
Femoral	Good	Death (Myocardial infarction)
Femoral	Failed after 2 years	Good
Femoropopliteal	Good	Death (Myocardial infarction)
Aortic-femoropopliteal	Distal portion failed	Good

to the femoral endarterectomy. Three patients underwent aortic-iliac-femoral reconstructions; all three retained their reconstructed segments, but two had later evidence of femoropopliteal disease.

Ten patients were followed up for five to seven and a half years. (Table IV.) Four patients in this group originally had pure aortic and iliac or isolated iliac lesions. In all four the operated segment has remained patent, but one patient has shown a distal femoral occlusion.

Four patients had pure femoral obstructions. Two have maintained excellent distal pulsations, but in one of these an aneurysmal dilatation of the femoral segment developed and a short portion was replaced with a nylon

TABLE IV
FIVE- TO SEVEN-YEAR FOLLOW-UP STUDY OF TEN PATIENTS

Site	Local Result	General Result
Four aortic-iliac lesions	4 Good	One distal femoral occlusion
Four femoral lesions	1 Good (but aneurysm treated successfully)	Good
	1 Good	Severe coronary disease (death from lymphoma)
	1 Improved	Stroke
	1 Late distal occlusion	Good
Two aortic-iliac-femoral lesions	Good	Myocardial infarct at five years with survival
	Good	Stroke, contralateral occlusion, died from heart trouble at seven and a half years

prosthesis. Normal pedal pulse is still present more than seven years after operation. One patient who retained patency of the femoral reconstruction had severe coronary disease and died five years after operation of a lymphoma. The third patient experienced symptomatic relief. His foot, which had been cold and pregangrenous, was warm and pink. He could walk a quarter of a mile without difficulty. However, the patient subsequently experienced an almost incapacitating stroke. In the fourth, occlusion developed at the end of the operated segment without dramatic change in the status of the leg six years after operation.

Two patients with aortic-iliac-femoral reconstructions were followed up five and seven years, respectively. One survived myocardial infarction at the end of the five-year period, the other died suddenly almost eight years after operation after having contralateral femoral occlusion and a cerebrovascular accident. In both patients the local result was good.

Considering the patients who could be followed up for four years or more, the ominous prognostic importance of the involvement of the femoral artery is seen from the following summary. Among those eleven patients who had disease apparently restricted to the region of the aorta and iliac arteries, there was only one late local failure and only three serious vascular complications at other sites. Two of twelve patients who had femoral arterial involvement in any form, however, had late local failures and eight manifested serious vascular disease in other sites. There were no deaths in the former group, but three occurred in the latter. One of the three deaths was due to lymphoma but this patient also had severe coronary disease. These findings may perhaps be partially explained on the basis of the age difference between these groups. The patients who had aortic-iliac disease without femoral involvement averaged 50.8 years of age at the time of operation; those patients who had evidence of femoral involvement too had an average age at operation of fifty-eight years. This correlates with the figures reported by Hines and Barker [4] describing a survival rate of only 46 per cent of patients with the diagnosis of arteriosclerosis obliterans who had been followed up three years. Our better survival rate is indicative only of the exclusion of patients who are the greatest risks from surgical consideration.

There were no amputations at a late date in any of the thirty-eight patients who had initially successful results.

It had been our strong clinical impression, based on the larger series of cases that includes those patients operated upon more recently [5], that weight gain and failure to follow a proper dietary regimen was apt to be associated with a higher rate of local and distant vascular failure. In this small group no such trend could be identified. Nine patients who were followed up more than four years had no local or distant failure, and five of these had gained weight. Fourteen patients had trouble at one site or another, three of these (all of whom died) had serious disease of a distant vessel but remained well underweight, and only six were described as clearly overweight.

The protective role of sympathectomy upon the distal femoral tree, whether partially reconstructed or not, is hard to define in combination with reconstruction. Among the patients followed up for four years or more, five underwent sympathectomy, and only one has had a late femoral occlusion. Eighteen patients did not undergo sympathectomy, seven of whom had femoral failures between two and six years after operation.

If one attempts to compare the possible patient-years of successful reconstruction, one finds that in the patients followed up for three years or more there is an opportunity of 142 patient-years of successful reconstruction; 137 patient-years were actually achieved.

SUMMARY

The status of forty-four patients subjected to endarterectomy prior to December 1955 indicates that in those patients in whom an initial good result exists, long-term persistence of patency is to be expected. In only four of thirty-eight such cases there was loss of the operated segment; in only one of these this loss was above the inguinal ligament, and in only this one the loss was the complete length of the operated segment. The other three retained patency of the proximal portion of the segment. The late mortality rate, however, of nine of thirty-eight of the successful cases, and the involvement of other vascular beds in eleven of the twenty-three patients followed up more than four years indicates the seriousness of the generalized vascular status of these patients. The merit of any major surgical procedure in this group of patients must be considered

against this unfavorable background of diffuse disease, especially as it relates to reconstruction in the presence of femoral arterial disease.

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DISCUSSION

Edwin J. Wylie (San Francisco, Calif.): On the basis of a study somewhat similar to that described by Dr. Barker, we have attempted to define more clearly the indications to be used for thromboendarterectomy on our Vascular Service, University of California Hospital, San Francisco. I would like to present the conclusions of this study. From a review of our data it became apparent that postoperatively our patients could be separated into one of two broad categories, defined by the location and extent of the occlusive lesion, and determined by aortography, and surgical exploration. The first category, designated as "aortoiliac," included patients with lesions limited to the aorta and common iliac arteries. These patients had no observable arterial disease distally, with the possible exception of the first 2 or 3 cm. of the external iliac arteries. The second category included patients with lesions beyond the bifurcation of the common iliac artery in the external iliac, femoral, or popliteal segments. Many of these patients also had gross disease in the aortic and common iliac segments. We designated this entire group as "iliofemoral." The noteworthy differences between these two groups of patients and the results of operation in each group became readily apparent.

The mean age of onset of symptoms of patients in the aortoiliac group was forty-nine years. Collateral blood flow was adequate to remove the threat of loss of viability in the lower extremities. Nevertheless, the disability caused by claudication in these relatively young patients was sufficiently great to justify arterial reconstruction so long as the surgical risk could be minimized. The current operative mortality after thromboendarterectomy in this group is less than 2 per cent. Of 160 patients who have been observed for four to nine years postoperatively, recurrent thrombosis of the operative area developed in only eight patients, all but one of whom have been successfully reoperated upon by thrombectomy or bypass grafting. The cause of late occlusion in at least five patients was the failure to remove all thickened intima distally at the time of the original operation. In three patients recurrent atherosclerosis appeared to be present in the endarterectomized segment.

We share Dr. Barker's interest in observing the relative failure of arteriosclerotic lesions to develop at other sites in these patients. Of the patients in the aortoiliac group who have been observed from four to nine years, only 14 per cent have shown evidence of significant arteriosclerosis at other sites.

The results of thromboendarterectomy and the subsequent fate of patients with lesions in the iliofemoral category were in striking contrast to the favorable experience with patients in the aortoiliac group. The mean age of onset of ischemic symptoms of patients with iliofemoral lesions was fifty-seven years. Although the occlusive lesions involved variable lengths of the arterial tree, aortography usually showed earlier lesions throughout the entire iliofemoral segment, often in the distal branches of the popliteal artery, and in the iliofemoral segment of the contralateral extremity. Of forty-five patients with lesions in the iliofemoral segment who survived for over four years after thromboendarterectomy, twenty-six had recurrent occlusion in the operative segment. This usually was attributed to progressive lesions of atherosclerosis proximal or distal to the endarterectomized segment. Continued patency was limited to patients whose initial lesions were only a few centimeters in length and in whom the remainder of the arterial tree was free from gross evidence of arteriosclerosis.

As might be anticipated in this older group of patients, development of atherosclerotic lesions at other sites occurred in a high percentage. By four years after operation, significant lesions had developed in coronary, cerebral, or contralateral arteries of the lower extremities in 50 per cent of these patients.

In view of the high incidence of recurrent or increasing atherosclerosis in the iliofemoral group of patients, it seems clear that the objective in arterial reconstruction must be considered in terms of short- rather than long-term success.

For patients with a simple thrombosis of the superficial femoral artery and a widely patent popliteal artery, disability is frequently limited to claudication and a walking tolerance of two to three blocks. For these patients we are currently proposing that arterial reconstruction of any type be deferred.

Disability is greater in patients with superficial femoral occlusion complicated by distal popliteal

disease. Unfortunately the presence of distal disease increases the likelihood of earlier failure following arterial reconstruction regardless of the method used. For these patients we suggest operation only if the viability of the limb is in jeopardy.

The greatest potential for improvement exists in patients with superficial femoral lesions complicated by complete or incomplete occlusions in the iliac arteries. Disability is great and the threat of loss of an extremity is frequent. We are currently using bypass technics with grafts extending from the aorta to the common femoral artery for extensive occlusions of this type, the objective being to provide increased collateral flow through the profunda. In a series of sixty-eight aortofemoral bypasses in the past two years, in which knitted Dacron® grafts were used, late failure from graft thrombosis has occurred in only one patient to date. Clinical improvement following a procedure of this type has usually been dramatic. Except for short occlusions of 1 to 2 cm. in the external iliac and femoral arteries, we have abandoned thromboendarterectomy for iliofemoral lesions except as a local procedure to facilitate the implantation of a bypass graft.

JACK A. CANNON (Los Angeles, Calif.): I would like to make two points.

We are really not obligated to treat patients according to roentgenographic findings. There is a tendency to do this. An obvious obstruction on a roentgenogram is most enticing to the surgeon, and yet it is astounding that the physician sees many patients who are sixty years of age and over with relatively warm feet, little or no claudication and no femoral pulses. This gives pause for thought and the application of surgical judgment.

The second point is that there is a tendency in present thinking to evaluate results in terms of restoration of pulse and the removal of a pressure gradient across a stenosis. There is more to the problem, however, because what we really need to know is how much the blood flow is increased in the distal tree as the result of the operation. Consequently, I believe that we must in the future pay a great deal more attention to the outflow tract and the amount of blood flowing into that tract both before and after operation.

Fortunately, excellent flowmeters are becoming available which can measure this factor. I recently operated upon a person who had a popliteal pulse and complete occlusion of the terminal popliteal artery. I was able to clean out the popliteal artery down to the posterior and anterior tibial vessels, leaving a strong bounding pulse. However, measurements of blood flow through the distal popliteal artery pre- and postoperatively were the same.

JOHN H. STEELQUIST (San Diego, Calif.): I would like to reiterate our belief about the preoperative diagnosis and the postoperative follow-up study of patients such as Dr. Barker has described.

Last year Dr. O'Connell and I described our use over a long period of time of the segmental plethysmograph and the digital plethysmograph in studying patients with arterial disease. We believe that we can determine fairly accurately those patients who have a significant amount of distal disease and in whom the distal run-off will be poor even after local endarterectomies are performed.

It is also possible, by studying digital flow, to evaluate fairly accurately the patients who will benefit not only by segmental endarterectomy, but also by concomitant sympathectomy to improve the total distal flow and relieve their symptoms.

We also find that this is an easy way to follow up patients after their endarterectomies and find out within a year or two or three, those patients who are beginning to have handicaps to their total flow because of the continued development of endarterial disease in sites beyond that of the original arterial surgery.

Dr. Barker should be congratulated on the fine results he has had. Dr. Connell and I have swung both ways. We started with the belief that endarterectomy was the best procedure. As satisfactory grafting material became available we were lured into the belief that bypass grafts would be highly satisfactory. However, since our number of unsatisfactory results from bypass grafts has mounted, we are going back to the procedure which Dr. Barker has described and believe that it is offering, in our hands at least, better results than the bypass procedure.

JOHN F. HIGGINSON (Santa Barbara, Calif.): I would like to ask what the authors' experience has been in the difficult group of patients they mentioned secondarily, those with involvement of the distal arteries, insofar as the use of anticoagulants is concerned. I have had encouraging experience using anticoagulant therapy through the immediate postoperative period and indefinitely thereafter. I question whether they have had similar experience in trying or using these drugs in an effort to prevent some of the late thrombotic episodes that perhaps are inevitable when you ream out some smaller vessels down to the tibials.

WILEY F. BARKER (closing): In answer to Dr. Higginson, we have not used anticoagulants extensively in the late follow-up period until recently. In recent times I have been inclined, with patients who seem to have this unfavorable distal tree, to carry them on anticoagulants as long as they live. I cannot tell yet what the results really are. I would hope they are better.

Dr. Steelquist's work is interesting with regard to improving one's ability to choose a course and to know exactly how far to carry that course, par-

ticularly with regard to the addition of sympathectomy and other procedures.

In summary, I think that when it comes to a choice between operations, since the outlook for aortic and iliac lesions is relatively favorable and the mortality rate low, it does not matter too much whether you use a bypass technic or endarterectomy. This is not so with femoral lesions. We are pessimistic and certainly admit that things are not all simple in this area. It is questionable just how much you are entitled to do to a patient to attempt a restoration of flow which seems to be, under present circumstances, only a temporary restoration.

One last comment regards something Dr. Cannon said concerning a problem that I am facing now. I have a female patient about sixty-five years old who has a lesion in the femoral artery, which as far as x-ray study shows is correctible. Actually her foot is warm and pink and claudication occurs after walking a distance of about 250 or 300 feet. I am not treating this patient because until about a year ago, when this claudication developed, she was instead suffering angina at this distance, and I believe that it is probably better to have claudication of the leg than of the myocardium.

The Management of Injuries to Major Veins

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ALTHOUGH a great deal of attention has been paid to vascular trauma, most of the emphasis has been on arterial injuries rather than on venous injuries. In a series of 228 patients with vascular injuries, we reported [1] fifty-two injuries to major veins. These proved to be of sufficient interest and importance to warrant a separate report.

MATERIAL

The fifty-two major venous injuries occurred in fifty-one patients, of whom forty-six were males and five were females with an average age of twenty-nine years. Sharp instruments accounted for twenty-three of the injuries, missiles caused twenty others, and blunt instruments accounted for nine. Twenty-nine patients had associated arterial damage.

The vein most frequently injured (nine times) was the superficial femoral. The internal jugular and the inferior vena cava were each involved eight times. The brachial was injured in seven cases, but the axillary only once. The popliteal was involved in four instances. Other major veins injured sporadically were the external iliac, the innominate, the hypogastric, the common iliac, the common femoral, the external jugular, the superior vena cava and the portal.

MANAGEMENT AND RESULTS

Simple ligation of injured veins is the classic method of management. Twenty-seven wounds were treated in this manner, including all the completely severed veins. Of the ligations, twenty-one were successful. Three patients required amputation because of associated arterial damage, two died of associated injuries, and one of a fat embolus.

Ten partially severed veins were repaired by lateral suture. Nine of the suture repairs gave good results, and one patient died of associated injuries.

In fifteen instances the venous defect was not corrected, either because an operation was not performed or because of failure to recognize the venous wound at exploration. Only two of these patients showed good results. Four patients died of venous trauma alone, and two of vascular damage plus associated injuries. Of the nine others, arteriovenous fistulas developed in seven, all of which were repaired later.

Six patients underwent sympathectomy because of concomitant arterial injury. Anticoagulants, usually heparin, were used in only eight patients. One-half of the patients required vigorous treatment for shock prior to operation.

Twenty-seven patients recovered without sequelae. Causalgia developed in two, probably due to associated nerve injury. Three cases of thrombophlebitis developed caudad to a ligated vein, one in the inferior vena cava and two in the superficial femoral vein, but no thrombophlebitis occurred after lateral suture repair. One man had postoperative hemorrhage from a ligated internal jugular vein, but he recovered.

Total mortality was ten (19.6 per cent). Four deaths were due primarily to venous trauma. One hospitalized patient stabbed himself in the brachial vein and died within two hours of injury before surgery was attempted. Two patients died of wounds to the innominate vein, one caused by a bullet and the other by a knife; neither was operated upon. The patient with the gunshot wound entered the hospital two hours after injury and died two hours later. The patient with the stab wound entered the hospital four hours after injury and died sixteen hours later. On the basis of auscultatory findings he was thought to have an arteriovenous

fistula, so an operation was not performed. The fourth patient who died, an eight year old girl, suffered a blunt injury to the abdomen in a school bus accident in which the hepatic veins were torn from the vena cava; she died while repair was being attempted.

The other six patients died of severe concomitant injuries. The veins injured were the inferior vena cava in two cases, the superficial femoral in two cases, and the external iliac and popliteal in one case each. The two patients with injuries to the inferior vena cava had gunshot wounds. In one, the bleeding from the inferior vena cava was controlled by Oxycel® and pressure, but the patient died two days later, probably from further hemorrhage. The patient who died of a fat embolus had sustained a severe compound fracture involving the popliteal vein.

COMMENTS

The mortality rate of nearly 20 per cent underscores the fact that major venous injuries cannot be dismissed lightly. While the high mortality rate in the cases reported was due in six instances to associated injuries to the head, chest, abdomen and arteries, four of the ten deaths were due directly to the venous injury. The over-all mortality rate compares closely with the mortality rate (17.6 per cent) in our series of 220 vascular injuries of all types. In that series, seven (43.7 per cent) of the sixteen patients whose deaths were related to the vascular injury were not operated upon. In the present series, four patients who died were not operated upon, and one patient's injury to the inferior vena cava was not repaired at operation. It is possible that these five deaths (one-half the total) could have been prevented by a more aggressive surgical attitude.

There is a tendency to give patients who are in shock large quantities of blood before taking them to the operating room. As in massively bleeding duodenal ulcers and ruptured aortic aneurysms, the best way to combat the shock is to stop the bleeding. This may require an aggressive approach to the major vessels of the neck, chest and abdomen, often under less than ideal conditions and frequently in the late hours of the night. The area behind the clavicles and sternum seems to be "no man's land." Because exposure is difficult without resection of all or portions of the clavicle or splitting of the sternum, there is a tendency to delay operation. Nevertheless, the surgeon must be prepared for these operations. It takes the greatest courage to desist from operating and the finest judgment to recognize the patient who will survive without operation.

Lateral suture repair should be done whenever possible. It was successful in every case and all patients who survived had good results. Ligation is usually satisfactory, but should be done only if lateral suture repair is not feasible. There has been a very low incidence of thrombophlebitis after ligation. Heparin does not appear to be of value and has some obvious undesirable effects in massively traumatized tissue.

When ligation is done in the inferior vena cava or the vessels of the lower extremities, the patient should have the legs elevated during the postoperative period and should not get out of bed without elastic supports. We are convinced that vigorous postoperative care of the patient who requires ligation of the inferior vena cava will prevent the undesirable sequelae of this procedure.

Failure to accomplish ligation of major veins which have been injured produces very poor results. In this series there were eight arteriovenous fistulas due to the fact that surgical exploration was not attempted. Five other patients died when injuries to major veins were not controlled. Only two patients who did not have operative control of a major venous injury recovered without sequelae.

Anastomosis of avulsed veins was not attempted in this series. It is doubtful that it would be successful because of the low pressure in the venous system. For this same reason, homografts and vein grafts or prosthetic grafts have not been used. The reports of methods to bridge defects in veins are not encouraging.

The eight injuries to the inferior vena cava deserve special comment. No such cases were reported in a recent large series of vascular injuries [2]. Five injuries to the vena cava were due to gunshot wounds. Four were repaired, with survival of three patients and death of the other due to associated injuries. The bleeding from the inferior vena cava in the fifth patient was controlled at surgery with pressure and Oxycel, but this patient died two days after operation.

Blunt trauma accounted for two injuries. One patient survived repair of the vena cava. The other patient was the eight year old girl

with avulsed hepatic veins who was previously mentioned. There was one iatrogenic injury in which the inferior vena cava was severely lacerated during an abdominoperineal resection and was repaired, with survival of the patient. There were no injuries to the vena cava by stab wounds.

SUMMARY AND CONCLUSIONS

In fifty-two major venous injuries over a ten-year period at the Los Angeles County General Hospital, the veins injured most frequently were the superficial femoral, the internal jugular, the inferior vena cava and the brachial. Eighty per cent of the patients survived with good results.

The profound shock which frequently occurs with venous injury must be combated by prompt surgery to control the bleeding site. This necessitates an aggressive approach to the vessels involved whether they are in the neck, chest, abdomen or extremities.

Lateral suture repair is preferred to ligation. Postoperative anticoagulants are of little value, and postoperative thrombophlebitis is not a major problem after repair or ligation. Grafts to bridge venous defects are not advocated.

There were ten deaths (mortality rate, 19.6 per cent), four in patients who were not operated upon and a fifth from bleeding at the site of an improperly controlled laceration of the vena cava.

Eight arteriovenous fistulas occurred in patients who were not operated upon. Prompt surgery might have prevented this disturbing sequela.

The eight injuries to the vena cava were of special interest. Five of the patients survived. Repair of the vena cava with lateral sutures is preferred to ligation.

When major veins are injured they must be repaired promptly to control bleeding, avoid arteriovenous fistulas and prevent death.

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DISCUSSION

Victor Richards (San Francisco, Calif.): I certainly agree with everything that Drs. Gaspar and Treiman have said. There are a few additional observations that I would like to make and a few points I would like to emphasize.

First, I agree with the principle of "turning off the faucet to stop the leak," and I think that an aggressive attitude in these cases is quite justifiable. Certainly the bleeding point should be found and the vessel repaired.

I believe repair of the vessel is far preferable to ligation, particularly in vessels the size of the inferior vena cava or the superior vena cava. The patients that Dr. Gaspar treated were repaired by lateral suturing. I have not treated such patients, but I think I would be tempted to try end-to-end anastomosis in the vena cava. Even though the late results may be very good, I have seen some very poor late results in patients who have come to me after having vena caval ligations elsewhere.

The question of anticoagulants is a rather complex one, and I am not sure that the final story on this has been told. It might be valuable to use them in venous repairs. We have shied away from anticoagulants because of the difficulties in control and some of the complexities of measurement, but in the last two years there have been great improvements in this. In the next two years we will find surgeons using anticoagulants more and more extensively, probably in this group of patients.

The really outstanding advances have been the development of a new and simplified test for measuring all blood-clotting factors—the Owren test. This seems to be quite universally satisfactory in the few areas where it has been tried, and we have recently had a little experience with a mechanical blood clot timer which simplifies the procedure of taking samples and measuring the clotting time and total blood-clotting factors in which we are interested.

Perhaps one of the reasons Dr. Gaspar asked me to discuss this paper is the report we submitted about two years ago on the use of venous grafts in the superior vena cava. Dr. Gerbode did rather extensive research work in the laboratory on the use of venous grafts and suturing the vena cava into the heart. In general, many of these venous grafts tended to thrombose and clot.

However, we were encouraged by some of this work and did treat four patients who had superior caval obstruction with bypass grafts. Two of these repairs were effected with homografts and the other two later, with plastic substitutes. The two homografts that were put in for obstruction of the superior vena cava running from the innominate veins down to the right auricle—both were sutured into the right auricle—have remained open, with dramatic relief of the superior caval obstruction. One of the plastic grafts in the venous system closed; the other has been lost to follow-up.

Another interesting experience we had in the

venous grafting area was in one severe case of hydrocephalus. A Sleeter valve had been put in but the internal jugular vein thrombosed. We then operated upon the patient again, in association with our neurosurgeons, and ran the plastic substitute from the ventricle of the brain down into the right auricle. This bypass has stayed open for about seven months now.

In my opinion, the success of the graft is determined by the pressure head in the flow rather than the fact that it is in either the venous or the arterial system. Certainly, if there is an adequate flow with a vessel of normal size, I believe there is a possibility that a graft will stay open. I have not had occasion to use one in the inferior vena cava, but I certainly favor the reconstruction of both the superior and inferior vena cava in injuries to these vessels.

CLARE G. PETERSON (Portland, Ore.): One feature of venous injury has been revealed to us from a continuing analysis of deaths and complications on surgical services at the University of Oregon Medical School Hospitals and Clinics. In our last morbidity–mortality conference, which is a multi-service review of deaths and complications, we were reviewing an instance of arterial air embolism from the cardiovascular service. In discussing this problem, we carried the discussion to the venous side, and found that in reviewing air embolism as a complication of venous injuries, we were seeing approximately two or three fatalities a year. These occur on all services: on the neurosurgical service, as a complication of neurosurgical diagnostic procedures that are done in relation to the great veins with the patient in the erect position; on the head and neck service, due to jugular vein injuries; in cardiovascular and thoracic surgery; following retroperitoneal air studies; from the pediatric surgery service, where a recent incident occurred in relation to a tear in the hepatic vein during attempted hepatic resection. On surgical services, there are aspirations of air into the venous system from a variety of sources. The anesthesiologists are acutely aware of this danger. It is a frightening thing to realize that as little as 1 ml. of air introduced directly into the venous system may produce an audible murmur, and that the pulmonary air lock which occurs may do so with catastrophic rapidity. Death from air embolism is a combination of mechanical block and reflex effect. A pulmonary artery air lock, cerebral or coronary artery air embolism may occur, and we have seen in most instances of venous air embolism a combination of emboli on the arterial side of the circulation, as well as the pulmonary air lock.

The death rate from air embolus of a surgical type from all services is appreciable following accidental tears of venous structures and a variety of diagnostic procedures which introduce air directly into the venous circulation.

ALOIS E. MOORE (San Diego, Calif.): Dr. Gaspar has brought a subject to our attention which has not been published in the literature. I would like to mention a report that my associate, Dr. Steelquist, presented about three years ago on postoperative care following ligation of the inferior vena cava, that is, femoral thrombectomy.

We have used this procedure in several patients. It was originally Dr. Steelquist's idea, and it seems that following ligation of the inferior vena cava thrombi will invariably develop in the deep venous system, that is, the femoral vein. The treatment is to open both femoral veins under local anesthesia twenty-four hours after ligation. One will find an organizing thrombus which completely fills the vein. This can be removed very easily through a small opening in the vein and blood flow established.

One patient wore elastic bandages for a year. He has required no pressure support of any kind for some four years, and the extremity is free of edema. I think that one should always consider performing this procedure twenty-four hours after ligation of the inferior vena cava.

LEONARD D. ROSENMAN (San Francisco, Calif): There was one aspect of venous injury which Dr. Gaspar alluded to which I would like to illustrate, that is, compression of major veins causing serious disability shortly after injury.

A fifty year old left-handed house painter suffered fractures of the upper six ribs and of the middle and lateral portions of his clavicle in an automobile accident. About ten days after this injury, during which time he was treated aggressively for his hemothorax and the respiratory embarrassment associated with it, massive dilatation of the superficial veins of his arm suddenly developed. These veins were obscured by a tremendous edema which extended from the finger tips to the shoulder.

At that time, it was thought that this man's career depended on his ability to use his left arm.

We simply resected the lateral half of his clavicle and exposed the dense fresh callus which surrounded the lateral portion of the subclavian and medial portion of the axillary vein. Over a distance of approximately 4 inches we freed the vein, which was narrowed to less than 1 mm. in diameter by the dense encircling callus, and removed a strip of the confining tissues.

There was a very remarkable and quick recovery, and the edema subsided. I think this is one form of venous injury in this area which should have our attention.

MAX R. GASPAR (closing): We have not put grafts into veins as Dr. Richards has. I have speculated that if we get a severe injury to the inferior vena cava again, it might be handled by a reverse portacaval shunt. In other words, ligate the vena cava cephalad, where it must be ligated, and

then anastomose the caudad end of the cava to the side of the portal vein. This has been done in dogs, and it has been reported and is acceptable. It might work in the human vena cava which is badly torn up or partially avulsed above the renal veins.

I thought Dr. Peterson's comments were very interesting. We have not had that experience, although we may have had it and not recognized it.

We have practiced Dr. Steelquist's suggestion about thrombectomy following ligation of the inferior vena cava with good results. However, I do not believe we have used it bilaterally. The fresh thrombus usually occurs on the side in which there was no thrombophlebitis prior to the vena caval ligation, and that is where you can get the best results.

We had a case somewhat similar to Dr. Rosenman's. It was a crush injury to the superior vena cava with a superior vena caval obstruction syndrome. This patient was not operated upon. He died shortly after injury.

Abdominal Aortic Aneurysms—Intact and Ruptured

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SURGEONS from Greek to modern times have sought an ideal operation for aneurysms, veritably a surgical Holy Grail. Antyllus, a second century Greek, described succinctly the nature and types of aneurysms and their treatment by ligation and evacuation. There was no substantial progress for 1,700 years although our surgical forebearers tried. Their roll call is an impressive one—Anel, Hunter, Dupuytren, Cooper, Halsted, Matas and many others. For the most part, they sought to occlude the blood flow through the aneurysm under attack. This was usually an aneurysm of the extremity, perhaps popliteal, femoral or brachial. The aorta, however, was not inviolate, for it was ligated by Cooper in 1817 in a near successful effort to control a ruptured iliac aneurysm.

It was not until this century that elimination of the aneurysmal sac with preservation of the blood flow was attempted. Matas made this effort using the patient's own tissues, a reconstructive or restorative aneurysmorrhaphy. Others turned to vessel transplantation, inspired perhaps by Carrell's success in the laboratory with the preservation and transplantation of tissues including blood vessels. Halsted, fifty years ago, replaced a popliteal artery sacrificed during the excision of a sarcoma with a segment of saphenous vein. About the same time Lexer excised an iliofemoral aneurysm, again using the saphenous vein to restore continuity. Shortly thereafter Bernheim and Halsted similarly treated a popliteal aneurysm prompting the former to report "The Ideal Operation for Aneurysm." His enthusiasm was not shared by Halsted who said in part: "Many surgical procedures have been called ideal and for their time have, perhaps, deserved this appellation. Many of them, however, were short-lived." He goes on to describe the hazard of thrombosis ensuing in the graft and extending into the artery with occlusion of important collaterals. To those of us who have implanted grafts into the popliteal region, this is by no means an outmoded concept.

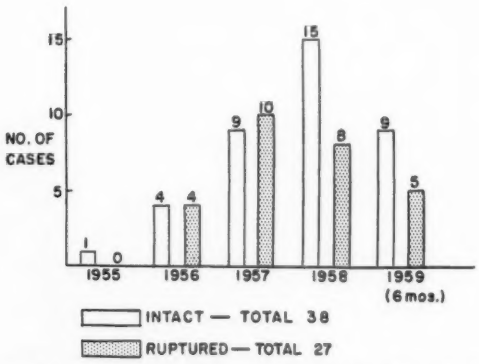

FIG. 1. Graph suggests a decreasing number of ruptured aneurysms excised and replaced with grafts as the elective ones increase (October 1955–1959).

It was not until this decade that resection of abdominal aneurysms became a reality. Robert Gross deserves much of the credit for stimulating clinical application of Carrell's work to major vessel surgery. In 1951 Dubost reported the first successful resection and replacement of an aneurysm of the abdominal aorta.

It was only after considerable work in the laboratory that we felt brave enough to proceed with the clinical series to be reported. The preliminary work entailed vessel preservation and transplantation in dogs, and procurement and preservation of human aortas. By 1955 we had established a blood vessel bank of freeze-dried vessels. In October of that year we resected and replaced our first intact abdominal aneurysm. In January 1956, we salvaged our first ruptured abdominal aneurysm. As luck would have it, both operations were successful although the

TABLE I
DATA ON THIRTY-EIGHT INTACT ANEURYSMS

Sex ratio (male: female)	7:1
Age (yr.) (mean age, 74 yr.)	51 to 84
Associated serious cardiovascular disease	50%

TABLE II
INTACT ANEURYSMS: SYMPTOMS

Symptom	No. of Cases
Pain	19
Mass	12
Pain and mass	4
Minimal or no symptoms	3

ruptured aneurysm gave us some trying moments. Thus encouraged by what we, too, hoped might be "the ideal operation," our series was launched.

In the past four years we have accummulated a modest experience with the surgery of abdominal aortic aneurysms. The intact and ruptured aneurysms differ so markedly that they are best considered separately. Our experience with the two groups has been nearly equal in size. We hoped that as we treated more elective aneurysms, we might see fewer ruptured ones, and this seems to be materializing. (Fig. 1.) The cases reported include a number of Dr. J. A. Elliot's; I am grateful to him for allowing me to include them.

INTACT ABDOMINAL ANEURYSMS

The Patient. We believe that any patient who has an unequivocal aortic aneurysm and the physical and mental vigour to enjoy life is a candidate for surgery. We do not believe that bedridden, confused, grossly senile patients are suitable candidates. This by no means eliminates many people with serious disabilities. (Table 1.) The profile of a typical patient is a seventy-four year old man with a history of at least one and probably more of the following conditions: coronary insufficiency with an earlier myocardial infarct, symptomatic hypertension and chronic respiratory disease. In addition, of course, are the miscellaneous conditions with which this age group are afflicted: hernias, prostatism and prolapse, to name a few. Happily, only one in eight will have associated occlusive vascular disease of the lower extremities.

The Aneurysm. All the intact aneurysms were responsible for symptoms (although perhaps minimal in degree). These could be broadly grouped into pain or a mass. In one-half the cases pain predominated, while in one-third the presence of a mass was the main cause of complaint. (Table II.) The pain or discomfort complained of by the patient varied considerably in degree. At one extreme was the "acute aneurysm" in which there was a sudden onset of severe abdominal and back pain. The absence of shock made the probable distinction from rupture which was only established firmly by laparotomy. At the other extreme were patients with grumbling abdominal discomfort. Indeed, a few of the latter cases were diagnosed by the radiologist during the course of investigating these rather non-specific complaints. This proved somewhat embarrassing to the clinician (shall we say physician) for the diagnosis lay literally within his grasp.

There was great variation in the manner in which the patient became aware of a mass in the abdomen. In some it was a change in girth, while in others it was a source of disturbance during sleep; in two it was described as an untoward mobility of the umbilicus while buckling the trousers.

In all the patients with an intact aneurysm it was possible to palpate a pulsatile mass. A simple x-ray film of the abdomen (on which we shall comment further) was found to be helpful. At first aortograms were used routinely but were found to contribute little. For the past two years aortograms have been used only when there are specific indications, in particular: when the physical findings or flat plate suggest an aneurysm extending to a very high level; and when there is evidence of distal occlusive disease and the state of the "run-off" is suspect.

RUPTURED ABDOMINAL ANEURYSMS

The Patient. The group of patients with ruptured aortic aneurysms was comparable in respect to age, sex and infirmity to the intact group. Superimposed on this rather forbidding clinical background was evidence of an acute abdominal catastrophe. All patients included in this group had a recent frank extravasation of blood outside the wall of the aneurysm. An attempt at surgical excision and graft replace-

FIG. 2A. Lateral view of the abdomen of an obese patient which demonstrates the ruptured aneurysm. (FROM: ELLIOT, J. A., MCKENZIE, A. D. and CHUNG, W. B. Ruptured abdominal aortic aneurysms. *Surgery*, 46: 605, 1959.)

FIG. 2B. Anteroposterior view of same patient demonstrating clearly the outline of the aneurysm (arrows). (FROM: ELLIOT, J. A., MCKENZIE, A. D. and CHUNG, W. B. Ruptured abdominal aortic aneurysms. *Surgery*, 46: 605, 1959.)

ment was made in almost all patients diagnosed. (Table III.)

Those in whom no effort was made were truly hopeless, examples being a patient with diffuse prostatic carcinomatosis and an institutionalized patient who had been bedridden and senile for the previous three years.

The Aneurysm. The diagnosis of a ruptured abdominal aneurysm is usually not difficult for the experienced observer. It evidently does provide a problem for the inexperienced, for there was an average delay of twelve hours, and in one case a delay of fifty hours, before surgical consultation was sought. The patients universally showed evidence of having suffered a near mortal blow, for prostrating abdominal pain and shock were present. The latter often becomes well compensated and reassures the unwary examiner. A variety of local symptoms may present which are indicative of the direction of rupture and haematoma dissection. (Table IV.)

In almost all patients a large aneurysm, often progressively enlarging, was readily evident on physical examination. In two cases in which the patient was thick-set and findings were inconclusive, anteroposterior and lateral x-ray films of the abdomen proved most helpful. This can

TABLE III
TREATMENT OF THIRTY-FIVE RUPTURED ANEURYSMS

Treatment	No. of Cases
No surgery attempted	4
Surgery attempted but not completed	4
Aneurysms excised and replaced by grafts	27

TABLE IV
LOCAL SYMPTOMS OF RUPTURED ANEURYSMS AS RELATED TO HAEMATOMA DISSECTION

1. Back pain: Retroperitoneal dissection
2. Gastrointestinal upset: Mesenteric dissection
3. Gluteal pain: Pelvic dissection
4. Genital pain: Perirenal and ureteral dissection

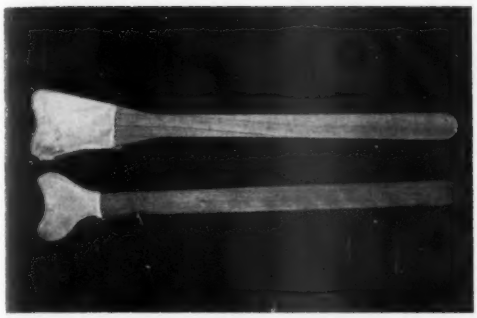

FIG. 3. Wooden aortic compressor with sponge rubber pads for emergency aortic control.

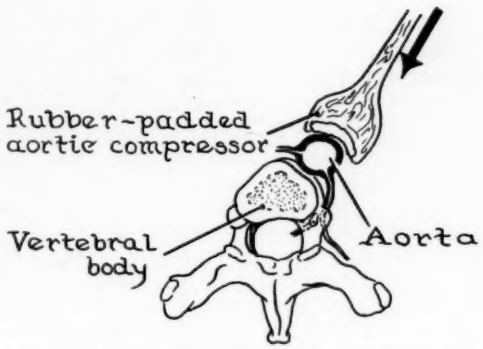

FIG. 4. Diagram representing the use of the compressor. (FROM: ELLIOT, J. A., MCKENZIE, A. D. and CHUNG, W. B. Ruptured abdominal aortic aneurysms. *Surgery*, 46: 605, 1959.)

be illustrated by one 290 pound patient whose x-ray film showed marked enlargement of a retroperitoneal mass previously identified on earlier investigation. (Figs. 2A and B.)

MANAGEMENT OF ABDOMINAL ANEURYSMS

Some initial reservations about the need for prompt all-out treatment of the ruptured aneurysm were soon dispelled. Two memorable patients, seemingly well compensated, went into profound shock while rather deliberate arrangements were being made. Since then, once the diagnosis is made, patients have been taken to the operating room area. No time is lost in preparing the patient, for the *sine qua non* of salvage is prompt control of the aorta proximal to the rupture.

In both groups the operation was conducted through a full length abdominal incision from the xiphoid to the pubis. Hypothermia has been used in all the more recent ruptured aneurysms and, indeed, for a number of the intact. We believe this has been advantageous with respect to shock tolerance and renal function, but do not have figures which would prove this belief. We have been able to and frequently do obtain control of the aorta well proximal to the aneurysm. This is of prime importance in the ruptured aneurysm and may be critical in any case if there is a misadventure. This control has been achieved by the use of "an aortic compressor" developed by one of our residents, Dr. Wallace Chung. (Figs. 3 and 4.)

The material used to replace the resected aorta has undergone progressive change. In the first half of the cases, homografts were used and in the second, synthetic materials. (Table v.)

There were several reasons for our change to synthetic materials. We found maintaining a sufficient homograft supply a nuisance and were disturbed by accumulating evidence in the literature of degenerative change. About the time better synthetic materials became available, the following experiences influenced us strongly to adopt synthetic materials: (1) In one patient, apparent early degeneration of the graft occurred with hemorrhage; a second graft again had to be promptly replaced. Finally, the insertion of a nylon prosthesis proved to be more than the patient could tolerate. (2) Rupture of the proximal suture line into the duodenum with exsanguinating hemorrhage was the cause of death in two cases of ruptured aneurysm, occurring thirty-three months and three months, respectively, after operation. (3) Atherosclerotic change of a severe degree had taken place in the homograft implanted thirty-three months previously.

TABLE V
REPLACEMENT MATERIAL USED IN SIXTY-FIVE AORTIC ANEURYSMS

Material	No. of Cases
Homografts	35
Synthetics	30
Teflon®	12
Dacron®	11
Nylon	7

RESULTS OF EXCISION AND REPLACEMENT

Intact Aneurysms. Several lessons were learned from the fatalities (seven postoperative deaths and one late death in thirty-eight cases).

Postoperative occlusion of flow to the lower extremities is an extremely grave situation. Two of the fatalities could be attributed to the need for a second operation to relieve obstruction. Every measure to avert this possibility is now exercised—the administration of heparin, and careful flush just prior to completing the distal anastomoses, firmly securing the distal intima and deliberate checking of the adequacy of flow to the lower extremities before the incision is closed.

Operation is not advised for patients who are crippled with other disease and are beyond rehabilitation. We now believe that barely correctable azotemia and angina decubitus, as found in one of our fatalities, constitute contraindications for operation. This patient passed no urine in his eighteen-hour survival and suffered a further infarct in the few shreds of myocardium left.

Careful and repeated evaluation of the patient's condition postoperatively is needed if shock is to be recognized and adequately treated. Two patients who died of this cause might have been saved.

Ruptured Aneurysms. Somewhat different lessons were learned from the morbidity and mortality in ruptured aneurysms (fourteen deaths in twenty-seven cases).

The wound will be subjected to severe strain from distention and coughing. Wound evisceration must be anticipated by a sturdy all-layer closure.

The high incidence of atelectasis and pneumonia due to retained bronchial secretions indicates the use of prophylactic tracheotomy in any patient in whom there is doubt about the ability to clear these secretions.

There is a need for immediate and painstaking fluid balance and administration, for in the majority of these cases, oliguria, even anuria, will prove to be a problem.

CONCLUSIONS

1. The history of the development of the modern replacement operation for abdominal aneurysms is briefly reviewed.

2. A series of seventy-three abdominal aneurysms, thirty-eight intact and thirty-five ruptured, seen over a four-year period is analyzed as to age, sex and associated disease of the patients. The symptoms of intact aneurysm have been found to be predominantly pain and, to a lesser degree, abdominal mass. Ruptured aneurysms were found to cause a general systemic reaction and, in addition, to have local symptoms related to the direction of haematoma dissection.

3. The abdominal route was used for the operative procedure in all cases with complete satisfaction. Useful adjuncts proved to be an aortic compressor to obtain proximal control when necessary and hypothermia which is thought to be helpful with respect to shock and postoperative renal function.

4. The replacement material used was human homografts in the first half of the series and synthetic material (with a developing preference for woven Teflon) in the last half of the series.

5. The results of operation indicate a decreasing mortality rate in both series but still an appreciable one: 18 per cent for the intact aneurysms and 50 per cent for the ruptured aneurysms.

6. It would seem that the search of the centuries for the ideal operation for aneurysm may be at an end. It is evident, however, that only too rarely do we see this condition in the ideal patient. Herein lies the main challenge of the present day surgery of aneurysms.

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DISCUSSION

K. ALVIN MERENDINO (Seattle, Wash.): This most interesting article has dealt with a problem of increasing importance as our aged population becomes proportionately greater. The philosophy regarding the indications and contraindications for surgery, the indications for aortography, and the progression from homografts to the synthetic grafts of Teflon and Dacron in general mirrors the general shifts in emphasis as surgical experience has accumulated. I can do nothing but indicate my agreement with these concepts in the management expressed by the author.

Dr. McKenzie has pointed out some of the symptoms of ruptured aneurysm and has indicated that the symptomatology may at times be misleading to the inexperienced. I am sure it is difficult for the experienced surgeon to believe that such can be the case. This fact, however, is well exemplified by a study of aneurysms made by Dr. Ralph K. Zech and myself some time ago (*Am. J. Surg.*, 20: 1150, 1954).

During the six-year period from 1946 to 1952, 3,313 autopsies were performed at the King County Hospital. An autopsy diagnosis of aneurysm of the aorta and iliac arteries was made in 116 instances (3.5 per cent). Of twenty-seven cases of ruptured aneurysms, (25 per cent) were operated upon with the mistaken diagnoses of perforated ulcer (four cases), volvulus (one case), urinary retention (one case) and pancreatitis (one case).

As additional support for the indications and contraindications for surgery which Dr. McKenzie presented, the average survival time of seventy-two patients, once the clinical diagnosis had been established, was nine months. This average period pertains to both ruptured and non-ruptured aneurysms. Undoubtedly, this average period of survival is shortened by the fact that almost 50 per cent of these patients were admitted with rupture and the clinical diagnosis was established for the first time. Of greater interest, however, is the fact that all patients, with one exception, died within thirty-seven months after the establishment of the diagnosis. Furthermore, 64 per cent died of causes other than aneurysms. This, I believe, emphasizes the fact, as presented by the author, that not all patients with aneurysms are candidates for excision. A large number die of other stigmata of arteriosclerosis, e.g., coronary thrombosis, cerebral apoplexy and renal complication. In general, when there are no important contraindications, I believe that the diagnosis of aneurysm is the only indication needed for surgery. It is also apparent that a careful analysis of the patient's assets and liabilities must be made to arrive at a sensible decision for his best interest.

The survival period, once symptoms relating to the aneurysm develop, averages 5.4 months. Once discomfort is present, either impending or actual rupture is implied, and there can be no alternative for surgery unless the patient is so debilitated or is dying of a related or different disease.

In 1955 I moved into the area of Teflon prosthetics. At that time, the unpurified dark brown fabric was used. One patient, who was operated upon in 1955 utilizing a seamed tube of woven Teflon, died in January 1960, five years after resection of the aneurysm and Teflon replacement of a coronary thrombosis. At autopsy, the graft was in excellent condition without evidence of calcification or any other complication. We also have a small colony of six dogs that have had Teflon replacement with the unpurified material. Three dogs have been sacrificed at five years without evidence of any changes on gross examination. The remainder will be sacrificed in the next two to three months when the five-year period ends. I am hopeful that the present purified Teflon grafts will hold up as well.

JOHN F. HIGGINSON (Santa Barbara, Calif.): I have enjoyed this paper by Dr. McKenzie, and since discussion of troubles is so often helpful to me, I thought perhaps you would enjoy hearing of some of mine.

In 1957 I removed an ordinary aneurysm and inserted the old type of woven Teflon graft.

The patient was operated upon under hypothermia and did very well. About three and a half weeks postoperatively, abdominal pain began to develop. We were suspicious of this, since at the time of surgery we had to perform a proximal cuff-thromboendaortectomy because of disease found when the aorta was transected above the aneurysm. Considerable thrombotic and atherosclerotic material was removed. This meant, of course, that the graft was anastomosed proximally to a very thin, tenuous bit of aorta.

Eventually the patient died, and it was found that the thin cuff of proximal aorta had ruptured and developed a false aneurysm, which had eroded into the duodenum with massive hematemesis. Actually we were able to operate upon him again before he died. We re-did this anastomosis, not once but twice. We did it once and were trying to repair the hole in the duodenum when it tore again due to this poor piece of aorta.

In retrospect, this probably would have been better excised, and complete extension done above, substituting arms for the renals.

Another patient demonstrated the fact that we are fighting a generalized disease. He was operated upon two years ago for an ordinary abdominal aortic aneurysm, with insertion of a knitted Dacron prosthesis. He did very well for three months when suddenly one evening he became unconscious. He died on the way to the hospital.

Although the abdominal aortic restoration was fine, there was present in the brain a large aneurysm of the right middle cerebral artery (obviously present but unsuspected for years), which had ruptured and caused his death.

In addition he had a rather rare aneurysm of the descending branch of the left coronary artery, so that he demonstrates that multiple aneurysms, not only of the aorta but also of other arteries of major significance and location, may be present in the same patient.

Finally, Dr. McKenzie mentioned that he was always able to control the aorta through the abdomen. A third patient, however, demonstrated that occasionally one may be confronted in the middle of the night by a patient with a ruptured aneurysm which extends from the iliacs up to and past the diaphragm into the chest, involving all abdominal visceral branches.

The left costal margin and diaphragm were cut, and the incision extended to control (as described by Merendino) the aorta in the chest. Mobilization was accomplished to the point of having the aneurysm laid bare when the patient died on the table. Incidentally, this man had an interesting anomaly which I had never seen before—complete absence of the celiac axis.

ALLAN D. MCKENZIE (closing): I think that we are learning that there are some patients we cannot help. It rather reminds me of what Dr. Key of Toronto said in discussing some of these cases. He said that some of these people who reach the age of eighty-five with an asymptomatic abdominal aortic aneurysm are candidates not for operation but rather for congratulation.

Hypertension Caused by Fibromuscular Hyperplasia of the Renal Arteries

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FIBROMUSCULAR hyperplasia of the renal artery has been recorded as the cause of renal arterial stenosis and secondary hypertension in at least six instances in the medical literature. Leadbetter's historic case [1] was a five and a half year old Negro boy in whom hypertension was first discovered at the age of six months. Following the removal of a malfunctioning pelvic kidney, blood pressure returned to normotensive levels. Pathologic sections showed segmental stenosis of the renal artery in which "the lumen was almost filled by a mass of tissue made up chiefly of smooth muscle outlined by an elastic lamella. Leadbetter also described "a defect in the wall of the artery, at one level characterized by thinning of the three layers and complete loss of elastic tissue."

Poutasse [2] of the Cleveland Clinic reports finding narrowing of the renal arteries in two young hypertensive patients (aged fifteen and nineteen) caused by fibrous intimal proliferation, a process which was bilateral in one of the patients. McCormack [3] extended the Cleveland Clinic series to three in the abstract of an unpublished report in which he alludes to finding fibromuscular hyperplasia in the renal arteries of three young patients. (The sex of these patients was not indicated.)

Leiter [4] reports findings during autopsy of a hypertensive forty year old man which suggested similar scattered lesions in the arcuate and interlobar arteries within the renal parenchyma. DeCamp [5] reported on a twenty-three year old man with a one-year history of hypertension in whom unilateral renal artery stenosis was caused by fibromuscular proliferation of the media, a lesion which DeCamp believed to be congenital. Resection of the stenosed segment and simple arterial anastomosis resulted in a postoperative fall of blood pressure to normotensive levels.

The purpose of the present report is to describe three additional cases in which reversible hypertension was produced by fibromuscular hyperplasia of one or both renal arteries and to call attention to certain distinguishing features of this newly recognized arterial disease.

CASE REPORTS

CASE 1. A thirty-five year old white woman was admitted to the University of California Hospital in December 1958 with a nine-month history of chronic fatigue and headaches. Two years previously, during the third trimester of her third pregnancy, she was found to be hypertensive. Blood pressure returned to normal after delivery. Hypertension and albuminuria recurred nine months before admission during the second trimester of a fourth pregnancy. Hypertension persisted following delivery; systolic pressures varied from 210 to 248 mm. Hg. Transient episodes of blurred vision occurred during the month prior to hospital admission. Attempts to control the hypertension with chlorothiazide and mecamylamine had been unsuccessful.

Examination in the hospital showed blood pressures ranging from 160/110 mm. Hg to 220/138 mm. Hg. The eye grounds showed KW II retinopathy. The cardiac size was normal. An electrocardiogram was interpreted as being within normal limits. A systolic bruit was audible in the epigastrium and extended into the right upper quadrant of the abdomen. Peripheral pulses were normal. Excretion of phenolsulfonphthalein was 55 per cent in one hour. Ptosis and a slight (2 cm.) decrease in the size of the right kidney were noted on otherwise normal excretory urograms. A Howard

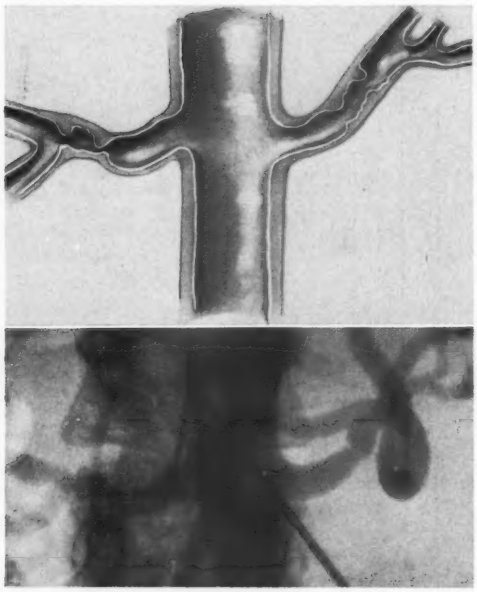


FIG. 1. Case 1. Translumbar aortogram and artist's sketch showing irregular stenosing lesions in the distal portions of both renal arteries.

divided urinary excretion study showed the following:

	Right Kidney		Left Kidney	
	Volume	Concentration	Volume	Concentration
20 min. PSP....	15 cc.	1 %	40 cc.	30 %
Sodium.....	8 cc.	1.5 mEq./L.	74 cc.	18 mEq./L.
Chloride.....		3 mEq./L.		25 mEq./L.

Renal arteriograms (Fig. 1) showed stenosing lesions involving the middle third of both renal arteries, and a seemingly greater degree of constriction on the right.

In view of the evident decrease in function of the right kidney, operation was undertaken with the aim of improving blood flow to the right kidney and thereby reducing blood pressure. The stenosing lesion in the right renal artery was palpable as a zone of unusual meaty thickening of the arterial wall. The pulse volume in the distal portion of this artery was noticeably weakened. Neither induration nor a pulse-pressure gradient was palpable in the left renal artery. The stenotic segment of the right renal artery was excised. Arterial repair was effected by simple end-to-end anastomosis of the divided ends of the renal artery.

Blood pressures during the immediate postoperative period varied from 120/88 mm. Hg to 140/95 mm. Hg. In the following fourteen months the maximum recorded pressure was 150/105 mm. Hg. The patient has had no further headaches or visual disturbances.

Howard studies performed three months after operation showed the following:

	Right Kidney		Left Kidney	
	Volume	Concentration	Volume	Concentration
15 min. PSP....	175 cc.	25 %	50 cc.	10 %
Sodium				
1st determination.....	73 cc.	44 mEq./L.	25 cc.	27 mEq./L.
2nd determination.....	71 cc.	8.8 mEq./L.	26 cc.	5.5 mEq./L.
3rd determination.....	81 cc.	6.8 mEq./L.	24 cc.	4.8 mEq./L.

The lumen of the excised segment of the right renal artery was irregularly narrowed; at one point it was less than 1 mm. in diameter. No gross evidence of atherosclerosis was found. Pathologic sections were made at 2 mm. intervals, which showed marked fibromuscular thickening of the media and only minimal focal thickening of the intima, limited to the most distal section. (Fig. 2.) In most sections the internal elastic membrane was intact, but in one area (Fig. 3) there was a hernia-like outpouching of this membrane through the whole thickness of the media. There was a terminal saccular dilatation of this outpouching where it abutted the adventitia, and the elastic tissue surrounding the sac was disrupted at this point. Biopsy of both kidneys showed moderate thickening and hyalinization of some of the arterioles, and occasional hyalinized glomeruli, consistent with the diagnosis of mild arteriolar nephrosclerosis.

Comment: In view of later experience and the suggestive evidence of decreased function of the left kidney postoperatively, the wisdom of limiting arterial reconstruction to only the right artery may be questioned. The seemingly normal thickness of the wall of the left renal artery upon palpation is in contrast to the extent of involvement observed by arteriography. (A similar experience was encountered in the second case.) Should hypertension recur or function of the left kidney become significantly depressed, repair of the left renal artery will be considered.

CASE II. A thirty-six year old white woman was admitted to the University of California Hospital

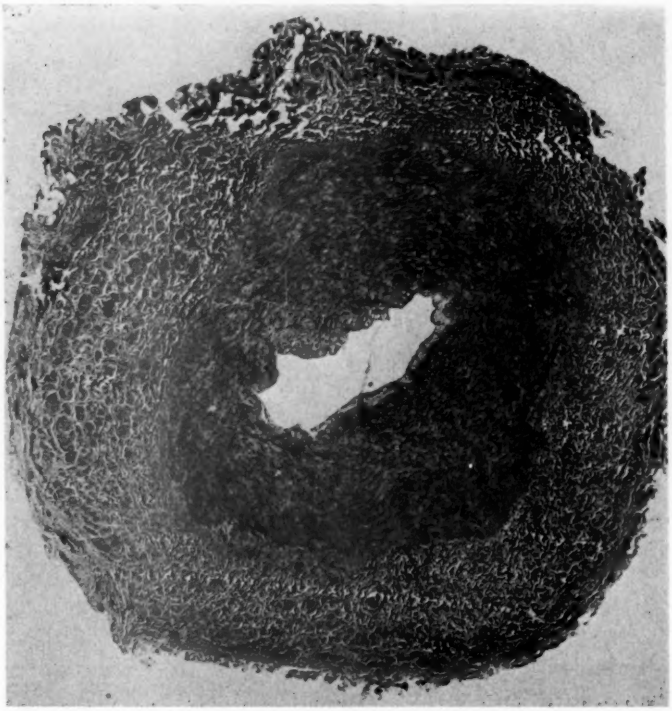


FIG. 2. Case 1. Cross section of right renal artery at point of maximum medial thickening, demonstrating an arterial lumen of less than 10 per cent of normal caliber. Hematoxylin and eosin, original magnification × 25.

in February 1959 for investigation of a possible lesion of the renal artery as a cause of hypertension. Her history showed that she had had hypertension, proteinuria and edema during her first pregnancy at the age of seventeen. Although these findings disappeared after delivery, transient hypertension recurred during each of three succeeding pregnancies over the subsequent ten years. Only after a fifth pregnancy at the age of thirty-one did hypertension become a constant finding. During the eighteen months prior to admission, blood pressures ranged as high as 250/140 mm. Hg. During this period severe headaches, repeated episodes of congestive failure, several attacks of angina associated with serial electrocardiographic changes of myocardial ischemia, and grade III KW eye ground changes developed. She required hospitalization twice for cardiovascular management and was treated with digitalis, diuretics and antihypertensive drugs. One week before the admission in February persistent severe pain developed in the left flank and lower abdomen.

On admission her blood pressure was 230/130 mm. Hg. A systolic bruit was audible over the epigastrium and over both costovertebral angles. A roentgenogram of the chest was normal. Urinalysis was negative. Excretion of phenolsulfonphthalein was 40 per cent in thirty minutes and 70 per cent in one hour. An intravenous pyelogram showed normal excretion of the contrast solution and a slight (1 cm.) decrease in the size of the left kidney. A Howard study of urinary function showed the following:

	Right Kidney		Left Kidney	
	Volume	Concentration	Volume	Concentration
15 min. PSP....	65 cc.	20 %	60 cc.	20 %
Sodium				
1st determination.....	22 cc.	9.7 mEq./L.	80 cc.	7.2 mEq./L.
2nd determination.....	63 cc.	9.3 mEq./L.	90 cc.	5.2 mEq./L.

Renal arteriograms showed stenotic lesions in the mid-portions of both renal arteries. (Fig. 4.) A zone

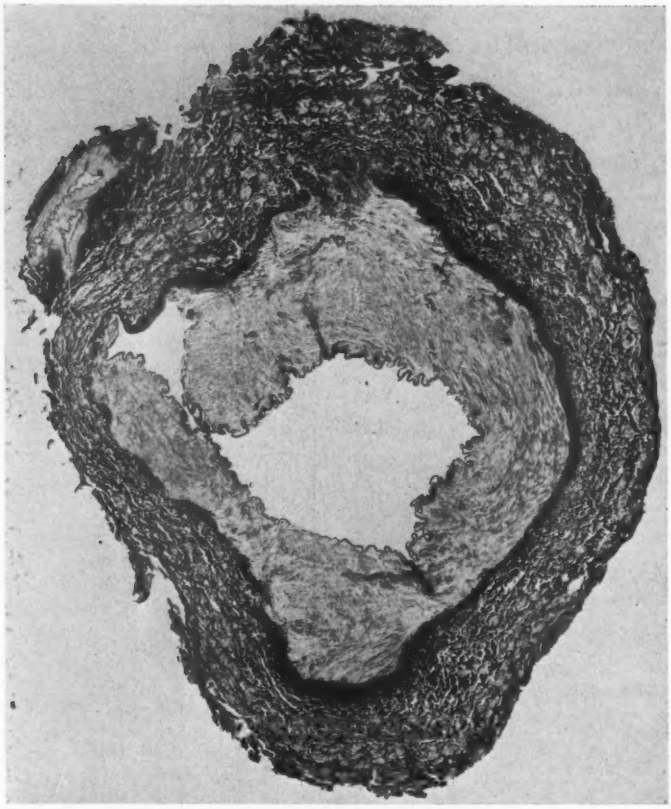


FIG. 3. Case I. Cross section of right renal artery showing thickened media and saccular outpouching of lumen. Weigert and van Gieson, original magnification × 25.

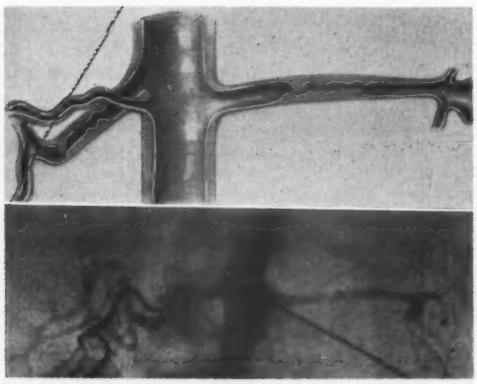
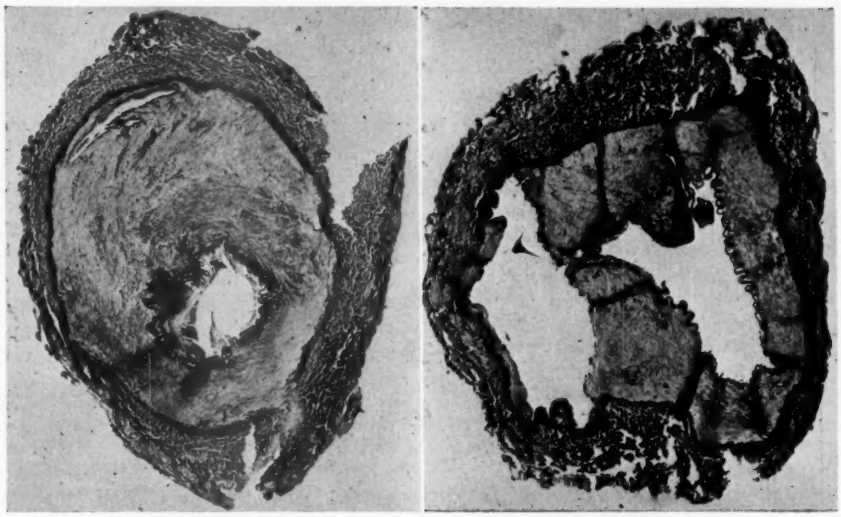


FIG. 4. Case II. Translumbar aortogram and artist's sketch showing "sausage-string" narrowing of the mid-portion of both renal arteries.

of incomplete vascular opacification of the lower pole of the left kidney was interpreted as representing segmental infarction.

Surgical exploration of the renal arteries on March 5, 1959, demonstrated that the vessels were of normal size but were palpably thickened in their mid-portions in the segment corresponding to the defect noted on arteriography. Pulse pressure was noted to be decreased upon palpation distal to the stenotic lesions. The thickened segment of each of the renal arteries was resected. Arterial repair was effected by end-to-end anastomosis of the preserved arterial segments. Ligation of a small arterial branch to the upper pole of the left kidney was necessary for control of hemorrhage.

Pathologic examination of the excised arterial segments showed irregular, sometimes eccentric, lumens which at some points were less than 1 mm. in diameter. Pathologic sections made at 2 mm.



5A 5B

FIG. 5. Case 11. A, cross section of right renal artery at the point of maximal intimal thickening showing that the decrease in caliber of the lumen is due primarily to medial thickening. Weigert and van Gieson, original magnification × 25. B, cross section of the left renal artery showing a small berry aneurysm with loss of elastic tissue in the wall of the aneurysmal sac. Weigert and van Gieson, original magnification × 25.

intervals showed marked fibromuscular thickening of the media and minimal focal intimal thickening. In a few areas the internal elastic membrane was fragmented and showed some duplication. (Fig. 5A.) Both arteries showed areas of outpouching of the internal elastic membrane similar to that seen in Case 1. (Fig. 5B.)

On the day after operation, blood pressure was 130/80 mm. Hg. and during the next two months it ranged from 140/80 to 150/95 mm. Hg. During this period the patient described marked improvement in all her preoperative symptoms.

In May 1959 anginal symptoms and headache recurred. The blood pressure rose to 180/120 mm. Hg. A Howard study at this time showed the following:

	Right Kidney		Left Kidney	
	Volume	Concentration	Volume	Concentration
15 min. PSP....	190 cc.	20 %	25 cc.	3 %
Sodium				
1st determination.....	149 cc.	6.3 mEq./L.	21 cc.	6.7 mEq./L.
2nd determination.....	234 cc.	5.5 mEq./L.	23 cc.	5.6 mEq./L.

A renal arteriogram showed a decrease in the size of the left kidney and stenosis at the site of the left renal arterial anastomosis.

Left nephrectomy was performed on June 10, 1959. Following operation the patient again experienced relief from her symptoms of angina, headache, malaise and congestive failure. Blood pressures in the following eight months have ranged from 119/70 to 140/80 mm. Hg without antihypertensive medication.

The resected kidney weighed 105 gm. and showed a segment of organizing infarction at the superior pole. The renal artery at the hilus was tortuous and of variable diameter; at one point the lumen was less than 1 mm. in diameter. A berry aneurysm, 0.7 cm. in diameter, was located 0.8 cm. proximal to the entry of the largest branch of the artery into the renal substance. Sections of this artery showed a thick muscular wall and substantially more intimal thickening than was present in the previously resected arterial segments. At the site of the aneurysm the media was markedly thinned or absent, and the internal elastic membrane could be traced into the distal wall of the aneurysm where it became fragmented and disappeared. (Fig. 6.)

In addition to the zone of healing infarction, the renal parenchyma showed a moderate degree of interstitial fibrosis and chronic inflammatory cell infiltration indicative of pyelonephritis.

FIG. 6. Case II. Cross section of a terminal branch of the left renal artery at the level of a small berry aneurysm. Note the disappearance of elastic tissue in the wall of the aneurysm. Weigert and van Gieson, original magnification × 20.

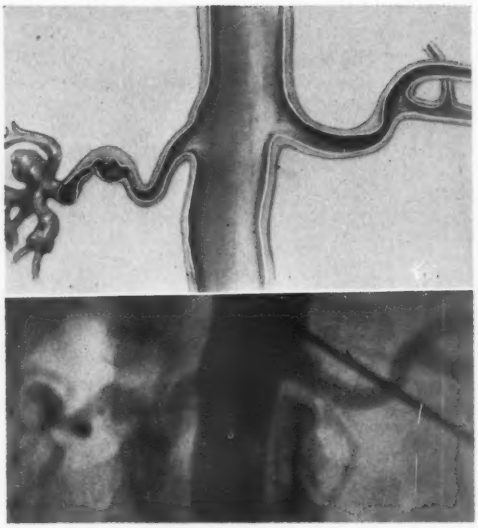


FIG. 7. Case III. Translumbar aortogram and artist's sketch showing irregular narrowing and focal aneurysmal dilatation of the right renal artery. Although the left renal artery was not explored, the small niche adjacent to the aortogram needle is interpreted as representing a segment of abnormal narrowing of the left renal artery.

The arteries and arterioles were thickened, with some intimal hyalinization of the arterioles consistent with arteriolar nephrosclerosis.

Comment: Although the comparative renal function studies of this patient prior to the first operation showed depressed function of only the left kidney, the clear evidence of bilateral occlusive arterial lesions prompted the decision to attempt reconstruction of both renal arteries. The recurrence of hypertension after a short normotensive postoperative period appears to have been caused by persistent or recurrent ischemia of the left kidney. Although this may have been attributable in part to the ligation of a branch of the renal artery, pathologic study indicates that the arterial lesions, originally apparent only in the renal artery, extended into

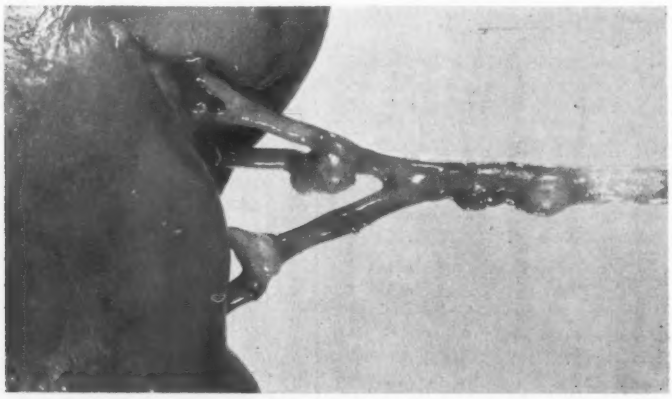


FIG. 8. Case III. Resected right kidney and attached renal artery showing focal aneurysmal dilatation in the renal artery and its terminal branches.

the terminal arterial branches in the renal pelvis, and likely had caused distal occlusion sufficient in degree to produce infarction in the lower pole of the kidney.

CASE III. A thirty-seven year old white woman was admitted to the University of California Hospital in April 1959 for investigation of a four-day episode of paresis of the left facial nerve. In addition to facial motor weakness on the left, she also complained of dysarthria, facial hypesthesia and blurring of vision in the left eye.

One year before entry, during the second trimester of her third pregnancy, hypertension and albuminuria developed for the first time. Two months after delivery her blood pressure was said to have increased to 240/140 mm. Hg. During this period she complained of fatigability, headaches and exertional dyspnea.

On examination, her blood pressure was 228/136 mm. Hg. The heart was of normal size. A systolic bruit was audible in the right flank and in the right upper quadrant of the abdomen anteriorly. Neurologic examination showed depression of left fifth and seventh nerve function. Albuminuria was noted on two of five urinalyses. Excretion of phenolsulfonphthalein was 30 per cent in fifteen minutes and 60 per cent in one hour. Intravenous urograms showed normal excretion of contrast medium from both kidneys. The right kidney appeared slightly smaller (2 cm.) than the left. Howard studies showed the following:

	Right Kidney		Left Kidney	
	Volume	Concentration	Volume	Concentration
15 min. PSP....		2.5 %		20 %
Sodium.....	6.5 cc.	1.1 mEq./L.	28 cc.	7.6 mEq./L.

Renal arteriograms (Fig. 7) showed sausage-string irregularities in the lumen of the distal half of the right renal artery and apparent tortuosity of its terminal branches. A small niche was observed in the mid-portion of the left renal artery.

On May 8, 1959, surgical exploration was performed. The wall of the distal half of the right renal artery was palpably thickened in an irregular fashion with alternating areas of thinning and aneurysmal dilatation. Similar changes were observed in the terminal branches of the artery just proximal to their point of entrance into the renal parenchyma. In view of the extensive involvement of the renal artery, it was apparent that arterial reconstruction would not be possible. The right kidney was resected. The resected specimen is illustrated in Figure 8. The left renal artery was not explored.

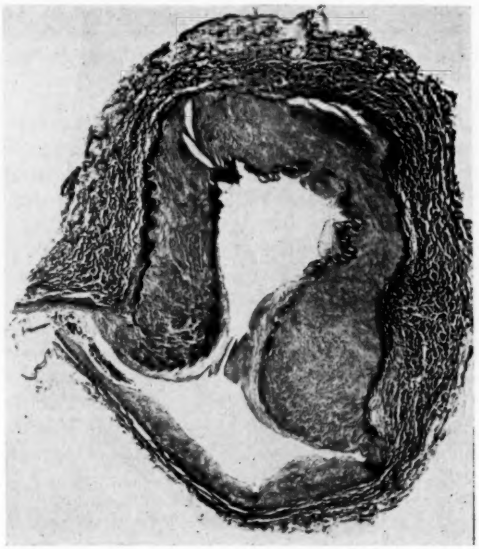


FIG. 9. Case III. Cross section of the right renal artery at the level of the aneurysm. Note the thin wall of the aneurysm and the intimal proliferation within the vessel at the point of communication between the artery and the aneurysm. Weigert and van Gieson, original magnification × 25.

The resected kidney weighed 125 gm. The 5.5 cm. segment of attached renal artery had three berry aneurysms, the largest of which was 0.6 cm. in diameter. One aneurysm was located in the main artery proximal to its first branching, and the other two were in the primary branches proximal to their point of entry into the kidney. Sections showed a small artery with a narrow lumen. The intima showed minimal thickening in scattered areas, and the media was markedly thickened by both smooth muscle and fibrous tissue. The internal elastic membrane was intact except in the areas of aneurysmal dilatation. On cross section the aneurysms were thin-walled, and herniation of the arterial lumen was seen through the media out to the level of the adventitia. The lining of each aneurysm consisted primarily of fibrous tissue; the normal smooth muscle and elastic tissue elements were either lacking or visibly reduced. (Fig. 9.) Some of the arteries within the kidney showed minimal intimal thickening. The kidney was otherwise normal.

On the day following operation the blood pressure stabilized at 110/75 mm. Hg and averaged 140/90 mm. Hg during the two weeks of hospital convalescence. The functional abnormalities of the central nervous system disappeared within seven

days. During the nine-month follow-up period the former symptoms attributed to hypertension have been absent. At the last recording, blood pressure was 115/70 mm. Hg.

Comment: The preoperative urinary excretion studies and findings at operation suggested that ischemia of the right kidney was the probable cause of hypertension. From the experience gained from the two previous cases and the observation of the defect in the left renal artery by arteriography, it was recognized that the basic disorder was potentially bilateral. Inasmuch as reconstruction of the left renal artery may eventually become necessary, operative exposure of the left renal artery was deliberately avoided at this time in order to prevent compromising a later procedure should the indications for operation develop.

COMMENTS

The uniformity of pathologic findings in the three cases described strongly suggests that the same arterial disorder had developed in all three women. Although the reports in the medical literature [*1–5*] would indicate that at least six other patients had been encountered with possibly the same pathologic entity, histologic description has been scanty.

The pathologic changes in all three cases present striking similarities. In all cases the histologic changes were confined to the renal arteries and their primary branches proximal to their entrance into the kidney. The proximal portions of the renal arteries appeared to be uninvolved. Hypertrophy of the media produced irregular and often extreme narrowing of the arterial lumen. van Gieson stains showed that this hypertrophy primarily involved the muscular tissue and, to a lesser extent, the fibrous connective tissue elements. The renal arteries in each case showed additional focal areas of medial thinning, disruption of both internal and external elastic membranes, and aneurysmal dilatation of the arterial wall. In two cases this produced grossly visible berry aneurysms, while in the third case (Case 1) this change was only seen on microscopic examination.

The intimal changes were limited to occasional areas of cellular proliferation, producing minimal focal thickening which was usually localized to an area adjacent to an aneurysm. The degenerative changes of atherosclerosis and the inflammatory reaction of the commonly recognized arteritides were notably absent. Significant findings in the renal tissue of all three cases were limited to the minimal nephrosclerotic changes characteristically observed in patients with hypertension.

The pathologic abnormalities observed in the renal arteries appear strikingly dissimilar to those found in atherosclerosis, thromboangiitis obliterans or the collagen diseases. Presumably the tendency for focal aneurysmal dilatation could be explained by the presence of degenerative or congenital defects in the elastic tissue. The cause of the unique muscular hypertrophy of the media remains unexplained.

The significance of the similarity in the clinical picture of these three patients can only be speculated upon at this time. It is noteworthy, however, that all three women were in the fourth decade of life and that hypertension had first developed in each as a complication of pregnancy. With the exception of possible coronary artery disease in one patient (Case 11), evidence of significant disease in vessels other than the renal arteries was lacking.

The antihypertensive effect of renal artery reconstruction or nephrectomy has been gratifying during the relatively short period of follow-up observation. Optimism, however, should be tempered by the observation that although depression of renal function was unilateral in two of the cases, arteriographic evidence indicated probable bilateral renal artery involvement in all three. This observation and the finding of extension of disease into the terminal branches of the renal arteries leaves the ultimate value of renal artery reconstruction open to question.

SUMMARY

Case histories of three hypertensive women in whom hypertension was apparently caused by the stenosing effect of fibromuscular infiltration of the renal arteries are described. Each of these women was in the fourth decade of life and had had hypertension during previous pregnancies. Blood pressure was restored to normal in each patient by either renal artery reconstruction or nephrectomy. The clinical and pathologic characteristics of this recently recognized arterial disorder are described.

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DISCUSSION

Maurice Sokolow (San Francisco, Calif.): The observations of Drs. Wylie and Wellington emphasize the resurgence of interest in renal ischemia as a cause of serious hypertension in man. It took 100 years for Butler to capitalize on Bright's observation in 1836 of the relationship between renal disease and hypertension, although Goldblatt's pioneer study in 1934 catalyzed the surgical triumph resulting from nephrectomy in Butler's case. The surge of enthusiasm which followed this first cure of hypertension by nephrectomy was tempered by Homer Smith in 1948, who accepted only 19 per cent cures in 262 cases of hypertension treated by nephrectomy; in a subsequent report of 575 collected cases in 1956 he increased the percentage to 26 per cent.* It is of interest that renal artery occlusion was not listed in his cases treated with nephrectomy, indicating that the lesion was missed. Although scattered cases were described previously, Poutasse and Dustan† in 1957 rejuvenated interest in renal artery occlusion when they found focal renal arterial disease in thirty of 104 selected hypertensive subjects examined by aortography over a period of two years. A more recent paper in 1959 described their experience in over 250 hypertensive patients who had had aortography. Twenty-five per cent were found to have focal renal lesions and significant improvement occurred in about 75 per cent of the operative cases.

The high frequency with which renal arterial lesions were found and the threefold improvement in their surgical results, compared to the over-all figures of Homer Smith, have naturally concerned all of us as to whether we were missing cases, when aortography should be done and whether nephrectomy or renal artery reconstruction was the procedure of choice. The three cases described by Drs. Wylie and Wellington might well have been overlooked some years ago, because the intravenous pyelograms disclosed no abnormalities and gross reduction of function of the involved kidney was not present.

Tables I and II define my concepts of the current indications for aortography in hypertension. Table III lists the advantages of reconstructive surgery rather than nephrectomy. In their paper, Isaacson

Table I
HISTORICAL INDICATIONS FOR AORTOGRAPHY IN HYPERTENSION

1. Recent onset of severe, persistent hypertension difficult to control in a patient with a negative family history of hypertension, especially if under thirty or over fifty years of age
2. History of recent severe pain in the flank or abdomen or trauma with or without hematuria, especially if there is a source for systemic emboli
3. Abrupt acceleration of pre-existing hypertension, especially in an older person

Table II
CLINICAL INDICATIONS FOR AORTOGRAPHY IN HYPERTENSION

1. Bruit over upper abdominal aorta, renal arteries or, less importantly, femoral arteries, especially in the presence of severe hypertension
2. Malignant hypertension
3. Unilateral decrease of 1 cm. or more in size of kidney, but not gross atrophy
4. Delayed excretion of dye in one kidney during excretory urography, expecially if normal retorgrade configuration
5. Evidence of calyceal atrophy rather than deformity on intravenous urography
6. Decreased water, sodium and PSP excretion in one kidney on retrograde study

Table III
ADVANTAGES OF RECONSTRUCTIVE SURGERY OF RENAL ARTERY AS COMPARED TO NEPHRECTOMY IN UNILATERAL RENAL ARTERY OCCLUSION

I. If the involved kidney is saved:
   A. More functioning nephrons remain
   B. The kidney distal to the obstructed renal artery is protected from hypertension and is physiologically more normal than the other kidney
II. Hypertension may persist if only the kidney with renal arteriosclerosis remains
III. Experimentally, hypertension of more than six months' duration in rabbits with clipped renal arteries may be reversed if the clip is removed, but not if the kidney is removed

* Smith, H. W. Unilateral nephrectomy in hypertensive disease. *J. Urol.*, 76: 685, 1956.

† Poutasse, E. F. and Dustan, H. P. Arteriosclerosis and renal hypertension. Indications for aortography in hypertensive patients and results of surgical treatment of obstructive lesions of the renal artery. *J. A. M. A.*, 165: 1521, 1957.

and Wayburne* illustrated the marked arteriolosclerosis in the opposite "unclipped" kidney of a nine year old child who died following nephrectomy for malignant hypertension due to localized occlusion of one renal artery near the aorta. The only arteriolar damage in the entire body was in the "unclipped" or opposite kidney, while the "clipped" or involved kidney which was removed was entirely negative histologically.

During the past two years we have seen about ten patients with severe hypertension caused by renal artery occlusion, most of whom have received striking benefit from surgery. Renal artery occlusion appears to be the most common cause of severe secondary hypertension, and should be considered a possibility in all cases of severe hypertension. Aortography is essential to establish the diagnosis and to delineate the anatomic features of the occlusion. Relief of renal artery occlusion is another of the many successes recently achieved by modern surgical technics.

WILLARD E. GOODWIN (Los Angeles, Calif.): Drs. Wylie and Wellington have not only presented an extremely interesting paper but also have clarified and accurately described this relatively unknown disease, fibromuscular hyperplasia of the renal artery.

It is interesting to trace the slow advancement of knowledge from the time of Goldblatt's original communication† in 1934, showing that hypertension could be produced by renal ischemia, to the authors' present study of these patients in whom renal ischemia was caused by fibromuscular narrowing of the renal artery. As mentioned in this paper, Leadbetter and Burkland recorded the first example of this entity in 1938.‡ Their patient, a child, was cured by nephrectomy. I was particularly interested in that case because I had the opportunity to review the end result showing cure of hypertension twelve years later.§ In the original paper they clearly show an illustration of a cross section of the renal artery containing a fibromuscular plug of tissue which is exactly the same disease that the authors report.

We have seen one similar case, now followed up for four years after surgery, at U.C.L.A. (UCLA No. 011-20-63). In the fall of 1955, a twenty-four year old woman, who was just a few weeks pregnant, appeared in the accident room complaining of a bee sting. A routine blood pressure recording showed that she had severe hypertension (200/110 mm. Hg). This observation led her to the urologic service where a prolonged and complete study finally revealed the cause of the hypertension. Intravenous and retrograde pyelograms were essentially normal, except for the fact that she had a calyceal diverticulum in the left kidney which contained a few small calculi. There was reduced function on the right side, however, and Dr. Winter's radioactive Diodrast® renogram demonstrated decreased vascularity and function of the right kidney. An aortogram clearly demonstrated a filling defect in the middle portion of the right renal artery. This was such an unusual lesion that the radiologists believed it to be an artifact, although the urologic service felt certain that this was the cause of her hypertension. Repeat aortograms again showed the lesion; thus the problem of management had to be discussed and decided.

At that time there were very few case reports of this condition treated successfully by conservative management and preservation of the kidney. The standard treatment for renal hypertension was then considered to be removal of the affected kidney. Because the patient had disease in the other kidney, it seemed desirable to preserve the right kidney if possible. After a very interesting consultation with Dr. Kattus (internist), Dr. Donald Burke (resident urologist) and Dr. William P. Longmire, we decided that an attempt should be made to preserve this kidney by removing the obstructing segment of the renal artery and reanastomosing it.

The operative diagnosis was "coarctation of the right renal artery," and on February 15, 1956, Dr. Longmire excised the narrowed segment and reanastomosed the renal artery. The patient's blood pressure promptly returned to normal and has remained so ever since. This is certainly the same disease which Drs. Wylie and Wellington have described so well in their patients.

It is of interest that although the blood pressure in our patient remains normal, follow-up studies with the radioactive renogram suggest a recurring obstruction. She is being watched closely because sometime in the future hypertension may again develop. Perhaps four years is too short an interval to know the final result in such a case.

It is of interest that our patient was pregnant when this was discovered, as were the authors' patients. I would like to ask Dr. Wylie if he thinks pregnancy can in any way be a contributing factor in this condition or is this a congenital disease?

EDWIN J. WYLIE (closing): We are currently studying two additional hypertensive women whose

* ISAACSON, C. and WAYBURNE, S. Malignant hypertension in a child due to unilateral kidney disease. *Arch. Dis. Childhood*, 32: 106, 1957.

† GOLDBLATT, H. L., JR., HANZAL, R. F. and SOMMERVILLE, W. W. Studies on experimental hypertension. I. The production of persistant elevation of systolic blood pressure by means of renal ischemia. *J. Exper. Med.*, 59: 347, 1934.

‡ LEADBETTER, W. F. and BURKLAND, C. E. Hypertension in unilateral renal disease. *J. Urol.*, 39: 611, 1938.

§ BURKLAND, C. E., GOODWIN, W. E. and LEADBETTER, W. F. A cure of hypertension by nephrectomy. *Surgery*, 28: 67, 1950.

renal arteriograms show the arterial irregularities we believe to be characteristic of fibromuscular hyperplasia.* Neither woman has been pregnant. It seems probable, therefore, that pregnancy is not a factor in the pathogenesis of this lesion.

On the basis of our present experience it would appear that this peculiar arterial lesion is a not uncommon cause of hypertension in young adult women. During the two-year period in which these cases were discovered and in which arteriography was used more frequently in the study of all hypertensive patients, there were only eighteen patients with the more common lesion of arteriosclerotic stenosis of one or both renal arteries.

My final comment concerns the distinct difference between the lesions of arteriosclerosis and fibromuscular hyperplasia. The histologic appearance of the latter lesion bears no resemblance to the histology of arteriosclerosis, regardless of the age of the patient. Furthermore, the typical lesion of arteriosclerosis, when it involves the renal artery, ordinarily does not extend beyond the first third of the artery. In the three patients presented herein and in the additional two patients currently under study, arterial narrowing involves the central or distal portion of the renal artery, sparing the proximal third.

* Since this paper was presented, these two women and three others have undergone successful renal arterial reconstruction for the treatment of hypertension. Of the entire group of eight patients, in only the first three had hypertension developed as a complication of pregnancy. The youngest woman in the series was eighteen years of age and the oldest was forty-two. The renal arteries of all eight patients showed the characteristic arteriographic deformity and the microscopic appearance of fibromuscular hyperplasia.

Synchronous Combined Abdominal and Perineal Resection of the Rectum for Carcinoma

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AFTER Davies described his method, many surgeons in Europe attempted to synchronize the abdominal and perineal phases of operations for resection of the rectum [1–3]. At this time, in several important hospitals in England and elsewhere in Europe surgeons employ that method. In America there has been less interest in the synchronous operation, and in the West there seem to be few surgeons who have had sufficient experience to evaluate the advantages or disadvantages of it. Hence we wish to report our efforts with the two-team operation, and to describe its features.

The combined abdominoperineal operation of Miles is well conceived to remove malignant growths of the distal colon and rectum. The surgeon cannot always easily perform careful, wide excision of the lateral areolar and muscular attachments within the pelvis, as Miles and others after him have advocated. The dissection in the depths of the pelvis may be hampered by the massive tumor, and hemostasis adequate for dissection under direct vision may be difficult to achieve. When inflammation blocks planes of dissection, a properly extensive resection is compromised, because the seeking after planes brings the surgeon too close to the rectum, or even through the friable wall into contaminated lumen. When he has completed the abdominal portion of the dissection he must thrust that portion of the colon to be resected beneath the pelvic peritoneum, a maneuver which gives him no security in what may happen when the mass below is extracted through the perineal incision. After the abdominal incision is closed, there are delays in preparing the patient for the perineal dissection. If the abdominal operation has been tedious, the delay further fatigues the surgeon, and his capacity for further surgery and his alertness are diminished. If the patient has not tolerated well the extensive manipulations or the loss of blood during the abdominal phase, the shifting of his body for exposure of the perineum may cause deterioration of his responses to the shocking operation. Also, of course, by performing the operation in consecutive phases, the duration of the procedure is lengthened.

Many operations for resection of the rectum have avoided all or some of the difficulties of Miles' procedure. They are, however, much less satisfactory as curative resections for cancer. In that category are the operations of Kraske and Hartmann. Another approach is the perineal abdominal sequence such as that proposed by Rankin. It commits the surgeon to total resection before he can assess the extent of the tumor or its metastases. A completely abdominal resection, as recommended by Fallis, is suitable only for non-malignant disease. In this era few surgeons have found it necessary to perform the abdominal and perineal portions as separate operations.

ADVANTAGES OF SYNCHRONOUS METHOD

Therefore, to gain the advantages of reduced operative time and reduced shock, two teams working together also improve the opportunities to dissect in the planes along the pelvic parietes by entering them from below as well as from above. The method provides for a clearer visualization of the lateral and posterolateral

portions of the pelvis, and facilitates accurate hemostasis in those regions. It encourages an unhurried and precise perineal dissection, and it permits inspection of the pelvic floor during closure on both its surfaces.

DISADVANTAGES OF SYNCHRONOUS METHOD

Some efforts to use two teams for resection of the rectum have been defeated by the problems of positioning the patient. Indeed, most of the disadvantages of the method come from the sacrifices the abdominal and the perineal surgeons make in accepting less than perfect freedom of motion and station. However, if the compromises cause only minor discomfort, and yield considerable advantage, then the method has much to commend it.

For example, the abdominal surgeon, who stands to the patient's left, will be displaced from the lower abdomen by a flexed thigh, and his left arm will be uncomfortable if the thigh prevents his own arm's free extension. However, it is possible to obtain excellent exposure without flexing the thighs more than a few degrees. (Fig. 2.) Similarly, the elevated thigh eliminates a position for the second assistant at the surgeon's left. We find a better position for the second assistant between the patient's abducted thighs. From that place he can retract in the depths of the pelvis and to either side with much greater ease than from the position he ordinarily occupies.

The perineal surgeon foregoes the freedom of the Kraske or the Sims positions, but relieves the patient of the traumatic and the shocking shiftings of the body that those positions require. The perineal exposure is modified from the usual lithotomy position. The surgeon sits as much below as between the thighs. Some flexion of the pelvis is provided by a sandbag under the sacrum, reducing the inconvenience caused by extending the thighs.

The duplication of surgical teams increases the number of persons in the operating room. A small room is not suitable as the increased population may cause confusion and disturbance. Both conditions may be avoided by planning and rehearsal; with experience there is less disorder and less hazard of contamination or other mishap. The requirement for additional personnel, both nurses and physicians, is a disadvantage which is offset by the time saved in a shorter operation.

CONTRAINDICATIONS

We have two contraindications for the synchronous operation. The tilt of the operating table may not be tolerable to patients with cardiac or pulmonary diseases; the tilt exceeds that used by most surgeons for the Miles operation, yet does not equal that of the original Trendelenberg position. The second contraindication is occlusive arterial disease in the legs. Too little concern is had for patients with that condition whose legs must be placed above the levels of their hearts during operations in lithotomy positions. If the total, available collateral circulation is dangerously small, new thromboses may occur when the legs are further deprived of blood during the time of elevation. Apropos of the foregoing, it should be recognized that the removal of the rectum, including the superior hemorrhoidal arterial system, from patients who suffer iliofemoral arterial occlusive disease probably deprives the legs of collateral circulation. We have observed one such patient in whom gangrene of the leg developed the day after his rectum was resected. Inspection of aortograms which show how the inferior mesenteric system enlarges to provide blood for the thighs would suggest that ischemic complications similar to that in the patient mentioned are more common than reported. At any rate, we believe it is contraindicated to raise the legs in those patients by stirrups for the time needed for the operation.

THE OPERATIVE POSITION

No unusual equipment is employed. The adjustable stirrups are available as standard items. (Fig. 1.)

Great attention is given to placing the patient in a stable posture, similar to that illustrated in Figures 2 and 3, after he has been anesthetized. He is moved toward the foot of the table, until his coccyx protrudes several inches beyond the break between the body and the leg portions of the table. The stirrups are secured to the table at angles of 135 to 150 degrees. The stirrup posts should be opposite and parallel to the axis of the outstretched legs.

Before the stirrups are rigged to the patient, a sandbag, 8 by 6 by 3 inches, is placed beneath the sacrum, well cephalad to the coccyx, so that the tip of the latter rides free above and beyond the break in the table. That maneuver is of signal importance to provide freedom for the

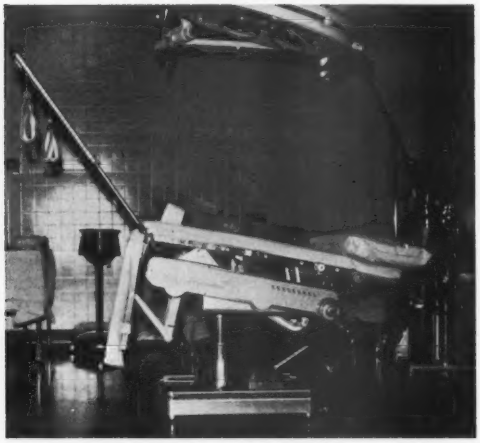

FIG. 1. Lateral depiction of the operating table and stirrups. The sacral sandbag is taped to the table.

perineal dissection. The sandbag must be supported if the table is designed with a triangular or trapezoidal defect in the caudal end of the body piece—most tables used for gynecologic operations have that sort of a cutout. One may correct that simply by placing a small board, like that used to hold the arm during intravenous infusion, across the defect in the table, and securing it with adhesive tape.

The feet are padded with sponge-rubber sheeting before the foot pieces of the stirrups are applied. We take care that the feet and legs swing free of the bars, and that they do not press against the angle of the stirrups.

The sacrum has been thrust ventrally by the sandbag, which lessens the lumbar lordosis. This reduces the 135 to 150 degree angle of the stirrups and causes only a 15 to 20 degree flexion of the thighs. The stirrups provide 45 degrees' abduction of the legs. Modification of the stirrups is possible to obtain additional abduction. We have not found it necessary.

The anesthesiologist places braces against the patient's shoulders, with care not to impinge on the brachial plexus. The braces share the burden of the body when the head of the table is tilted downward. When a tilt of about 20 degrees has been reached, the buttocks are separated by traction strips of waterproofed adhesive which are fastened to the table. A Foley catheter in the bladder is connected with sterile tubing which is led over the right groin to a bottle on the floor. In male patients the genitalia are drawn toward the right groin and held there with strips of tape.

The assigned nurse prepares the abdominal, pubic and perineal fields as one, beginning her scrubbing at the nipple line and proceeding toward the buttocks. The upper thighs are prepared as well as the posterolateral surfaces of the buttocks. We place sterile drapes beneath the buttocks to the level of the sandbag, and clip the drape to the skin just behind the tip of the coccyx. We place long drapes the length of the patient inside the thighs, and wrap the legs

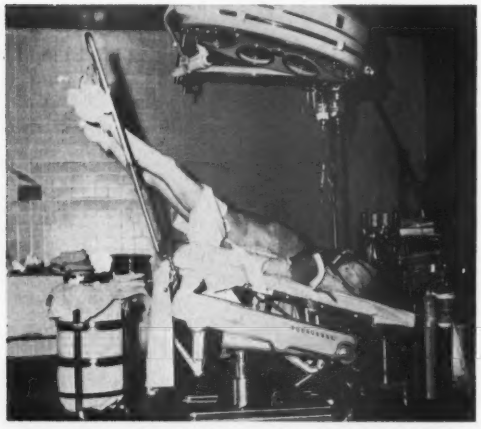

FIG. 2. The patient before draping. In most instances, the legs are more nearly parallel with the stirrup bars than is shown here.

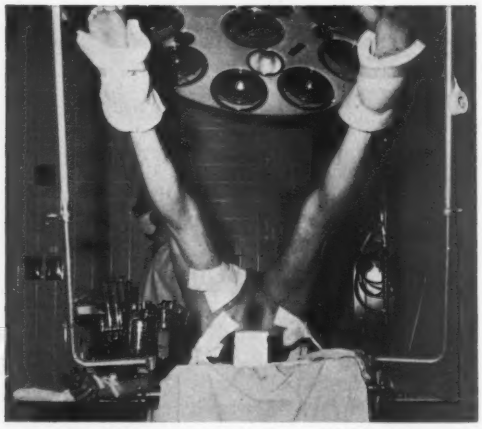

FIG. 3. The exposure of the perineal field is adequate. Note that the surgeon works beneath rather than between the outstretched thighs.

with the ends. With care, and a liberal supply of sheets and clips, the draping properly exposes sterile and protected fields. In essence, there is one field from the upper abdomen over the mons and to the coccyx. The head of the patient is draped beneath an instrument stand. The operating nurse has greater freedom standing near the patient's head, to his right, adjacent to the instrument stand.

The anesthesiologist works to the left of the patient and slightly behind and to the right of the surgeon, who is separated from him by drapes. The first assistant is opposite the surgeon. The second assistant stands between the patient's legs using a lift if needed. When the abdominal team no longer needs his assistance to expose the deep pelvic structures, he steps away to assist the perineal surgeon. The latter sits, his eyes at the level of the anus—somewhat lower than the ordinary lithotomist's position—and (as already noted) he works from below as well as between the legs. Because the perineal team expose their backs to the patient's thighs, to each other and to their operating nurse, it is wise to cover their backs with sterile gowns.

THE OPERATION

The abdominal surgeon begins the operation by marking the expected point of emergence of the colostomy through the skin of the left abdomen. If a colostomy is to be constructed, he will excise a disc of skin. A midline incision is opened from the pubis to above the umbilicus. The surgeon carefully explores the abdomen, and he may supplement palpation with coloscopy if there has been a suspicion of polyps proximal to the rectal tumor, anywhere in the colon. He displaces the small intestine from the lower abdomen. Restraining packs are seldom necessary because the viscera gravitate toward the diaphragm. The rectum and sigmoid are freed in order to determine the extent of the bowel below the tumor. Frequently we have observed that a lesion of the rectosigmoid, 5 or 6 inches from the anus, may present as a rectal tumor because it tends to intussuscept and to remain fixed in the pelvis. In such a patient the examiner cannot estimate the true level of the growth before surgery because the rectal folds are not straightened by the ascent of the tumor out of the pelvis ahead of the sigmoidoscope. Not until the rectum is dissected can he assess the tumor in terms of its position, size, penetration and regional metastases. Also, he cannot make the decision beforehand to perform a complete resection or to anastomose the upper sigmoid to the lower rectum after removing the tumor-bearing segment. If the latter course is taken, the perineal team may be dismissed. If the former is elected, the anus is closed at this stage of the operation.

The surgeon selects the level at which he wishes to divide the mesentery of the left colon, and he sections both the mesentery and the bowel. The proximal end is covered, allowing time for evaluation of its blood supply before it is used for a colostomy or an anastomosis.

When the abdominal team has freed the rectum down to the pelvic diaphragm, the perineal surgeon begins his phase of the operation, incising the perineum to remove an ellipse of skin with anus and ischiorectal fat. He enters the pelvis by incising the musculofascial tissues anterior to the tip of the coccyx. The two teams may assist each other to define the lateral extent of the pelvic diaphragm and to remove or preserve portions of the seminal vesicles, prostatic capsule, vagina, etc.

After the specimen has been removed, lights are directed into the pelvis from below and above. We irrigate the field of dissection with large volumes of warm saline solution which makes possible a more precise hemostasis than any other technic. This maneuver alone would commend the operation to us.

The closure of the pelvic peritoneum with interrupted and running sutures is inspected from below before the perineum is closed around several drains which lead into the presacral hollow.

It is our practice to suture the peritoneum of the lateral gutter to the colon, up to the colostomy. We fashion the colostomy, through the excised defect previously marked, by suturing the end of the colon to the skin. The colostomy should be constructed without angulation, to allow passage of an irrigating tube or an examiner's finger directly cephaled and dorsally along the descending colon.

EXPERIENCE WITH THE TWO-TEAM METHOD

We have prepared eighteen patients for the combined operation. In eight operations, anterior resection with anastomosis was performed; in the others, synchronous combined resections were completed.

We have had no mortality in the latter series

and no complications ascribable to the unusual position or drapes. In the entire group there have been no discomforts suggesting unusual strain in the knees. In one patient with chronic cervicitis, whose resection included a segment of posterior vaginal wall, an extensive infection developed in the perineal wound. One other patient had a less serious presacral infection. Two patients had superficial, small infections in the abdominal incisions. Another patient had recurrence of carcinoma in the pelvis after anterior resection. A synchronous combined resection was performed eighteen months after the first operation. We think the difficulties of the second operation were lessened by the synchronous approach.

SUMMARY

A technic is described for synchronous combined abdominoperineal resection of the rectum using two teams of surgeons and assistants. In our opinion, the advantages of the procedure outweigh the disadvantages. The few counter-indications to the method are discussed.

Acknowledgment: We wish to thank Dr. J. O. Smith for permission to include two patients treated by this method by him and Dr. Feigen.

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DISCUSSION

Robert A. Scarborough (San Francisco, Calif.): The authors have presented a good case for the synchronized combined abdominal and perineal resection, conceding certain objections and contra-indications to the procedure. I would like to voice further objections to this two-team operation.

I believe there should be one responsible surgeon who performs or personally directs the precise limits of adequate resection in each patient. For carcinoma of the upper rectum, the final decision between abdominal and perineal resection, and resection with pelvic anastomosis frequently cannot be made until wide mobilization of the rectum has been performed posteriorly down to the coccygeal fascia, anteriorly below the level of the seminal vessels and prostate, and laterally with division of the anterolateral ligaments. If it is then decided that the operation must be completed by posterior excision, this can be completed readily in five to ten minutes following closure of the abdomen.

When combined resection must be performed for carcinoma of the lower rectal ampulla, the success of the procedure is dependent to a high degree upon the adequacy of the widest possible perineal excision. This should certainly be the direct responsibility of the operating surgeon who, for this portion of the operation, should have the best possible exposure for the job.

I submit that this is not the case, and I quote from the authors' paper: "The perineal surgeon sits as much below as between the thighs."

The authors concede some disadvantage of the synchronous procedure in freedom of motion and station for the abdominal surgeon. I certainly agree with this. The positioning of a second assistant for the abdominal procedure, standing between the patient's abducted thighs, must certainly interfere with the perineal team working below and between the thighs.

The problem of exposure for dissection deep in the pelvis is certainly a real one, whether dissection is performed from above, below or synchronously from both directions; however, this particular problem cannot be circumvented until the bulky mass of the mobilized rectum is removed from the pelvic space.

I believe the major reason for the development of a synchronized operation has been the justifiable concern about the occurrence of shock when the patient is turned for the perineal phase of the procedure. In our experience this need not be of any concern. Our patients are turned to a prone position and there is no significant change in pulse or blood pressure. We credit this to the efficiency and alertness of our anesthesiologists. It is paramount that continuous blood replacement be maintained during the entire course of the operation, and it is customary to infuse quickly 150 cc. of blood just before the patient is turned, in anticipation of a probable sudden loss of a like amount while the perineal phase of the operation is completed, usually in five or ten minutes.

I certainly hope that Drs. Rosenman and Feigen will not be offended by this critical discussion. I intend no personal criticism, but rather wish to congratulate them on a most earnest effort to improve the technic of abdominal perineal resection.

Walter D. Birnbaum (San Francisco, Calif.): I am afraid that I am in sympathy with Dr. Scarborough's views, having attempted this procedure on several occasions. However, I can personally testify to the skill and high degree of integrity of the authors who presented this paper.

There is an additional advantage in positioning the patient in the manner described, even when a low anterior resection is being planned. This is a procedure which is now being performed almost routinely, at some of the centers in England particularly. By so positioning the patient, irrigation, either with a carcinogenic solution or copious lavage with plain saline, is possible.

The merit of such irrigation in lowering the recurrence rate of cancer in low anterior resections is becoming increasingly evident, so that in some centers where this is being done, again particularly St. Mark's, the recurrence rate has been lowered from a former 20 per cent to an almost negligible figure over a period of five years. Having the patient positioned as has been shown, and having the rectum available for irrigation, are points worthy of consideration.

ALLAN D. MCKENZIE (Vancouver, B. C.): I would like to support the authors' proposal of synchronous combined abdominal perineal resection. We have used this in Vancouver very extensively, but we do not use the position that was described. Rather, we use the Lloyd Davies stirrups as supplied by Down Brothers in London. We find these are very convenient and comfortable to work with.

We may not add a separate team; we may add one person only. Often the surgeon who is performing the abdominal resection does the perineal resection also. It really does not complicate the procedure. The exposure is excellent, and we have the advantage of irrigation that was mentioned. We save time and are satisfied that there is less shock to the patient. Our experience has been most satisfactory.

EDWARD N. SNYDER, JR. (Pasadena, Calif.): I support the authors' technic. This procedure has been used very extensively and successfully at the Los Angeles County General Hospital, only it is known by the less euphonious term of "two-teamed combined abdominoperineal resection."

At the County Hospital many of the patients are debilitated and elderly, and it has been my impression that they have been especially benefited by the use of the "synchronous technic," as contrasted with the conventional separate approaches.

Dr. Scarborough is a very dextrous surgeon who performs a considerable amount of radical surgery on the colon and rectum. Most of us are not so fortunate or perhaps so gifted as he, and I doubt if many of us can perform the perineal phase of the dissection in five to ten minutes.

I urge all of you to at least try the authors' technic before passing judgment.

LEONARD D. ROSENMAN (closing): With respect to the evaluation of the extent of the resection, the abdominal surgeon, of course, is the primary surgeon in the team as he makes the decision for resection. It is his responsibility; he has consultation with the perineal surgeon when the decision for or against anterior or complete resection is made.

The extent of the resection below, of course, is going to vary from surgeon to surgeon. In performing the two-team operation, one must have confidence that both the abdominal and the perineal resections are going to be adequate to the tasks.

The problem of shock has been more of a concern to us than to Dr. Scarborough, because we have had several patients who have had real difficulties during the turning phase; hence, we avoid it.

The position of the perineal surgeon is not difficult. He has very few limitations. His exposure is less limited than the abdominal surgeon's by the use of this technic, that is, the difficulties with position affect him less than they do the abdominal surgeon. This is a form of the lithotomy position which is superior, insofar as exposure of the posterior portion of his excision, to that which is usually obtained with the lithotomy position where exposure is gained only by marked flexion of the thighs.

By extending the buttocks beyond the end of the table and flexing the coccyx and the sacrum forward with the sandbag strategically placed, the exposure of the coccygeal area is improved. As has been stated by the other discussers, if anterior resection is elected, the establishment of perineal drainage posterior to the anus is a simple matter. The exposure is ideal for that.

The Mallory-Weiss Syndrome

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MASSIVE upper gastrointestinal hemorrhage originating from lacerations of the cardio-esophageal region caused by repeated vomiting was first described by Mallory and Weiss [1] in 1929. At that time they presented some fifteen cases which they thought represented typical examples of such hemorrhage. However, only four of these were verified either by surgery or by postmortem findings so that the exact etiologic factor in the other eleven cases must still be considered somewhat equivocal.

In 1932 the same authors [2] reported that they had observed six more cases which they thought belonged in the same category. However, they were able to confirm the findings in only two of these six cases. From that point on until 1953 there were no further reports in the literature. At that time Decker, Zamcheck and Mallory [3] reported proof at autopsy of eleven more cases of the same lesion from the same clinic. Since 1953 there have been some six articles which we have been able to find in the literature describing similar cases. However, all of these have been individual case reports.

Despite the obvious assumption that such a paucity of case reports would indicate that this is a very rare disease, it has been my firm conviction for some period of time that it has been a lack of awareness of such an entity on the part of the clinician, surgeon and pathologist that has resulted in failure to recognize the disease.

In my own experience I have encountered two definite, surgically proved cases of this syndrome, and it is my hope that I did not miss other cases in the past when I explored patients for massive gastrointestinal hemorrhage and no source of the bleeding could be demonstrated.

In their original report, Mallory and Weiss [1] were impressed by the past history of a large intake of alcohol plus violent vomiting and retching as predisposing factors prior to the onset of the massive bleeding. In their later reports, they were much less impressed by the necessary correlation of the alcoholic intake and became thoroughly convinced that the vomiting was the actual primarily important predisposing agent. This fact seems to have been confirmed by all of the subsequent reports, including my own, with vomiting being reported in all but one of the cases in the literature.

CASE REPORTS

CASE 1. S. O., a thirty-three year old physician, was admitted to the hospital on April 16, 1956, because he had vomited large amounts of blood. The patient had suffered with migraine headaches for years and stated that during one severe attack twelve years prior to this illness he had vomited blood. During subsequent attacks of headaches he had vomited on many occasions but never any blood. On the afternoon of admission he had a severe migraine headache with nausea and vomited a large amount of normal stomach contents around 6:00 P.M. At 7:00 P.M. he vomited a very large amount of fresh blood and clots, and fainted. During the ambulance trip to the hospital he again vomited considerable blood. There was no history of recent gastrointestinal distress of any nature.

The patient was treated by the ulcer regimen. A Levin tube was inserted, and fresh blood was obtained. Blood transfusions were started and during the night 7 units were given. A Sengstaken tube was later inserted and 2 pounds of traction against the gastric balloon failed to stop the flow of bright red blood.

His hemoglobin and hematocrit had continued at fairly normal levels but his pulse rose from 92 to 122 and remained in that range. After some six hours of constant observation and with bright red blood still coming almost constantly from the tube, exploratory laparotomy was recommended and performed. The preoperative diagnosis of laceration of the stomach secondary to vomiting was confirmed at surgery. A large 2½ to 3 inch laceration and a parallel 1 inch laceration were found with a spurting artery in the upper portion of the larger laceration. Treatment consisted of simple transfixion of the bleeding vessel and whipping over the two lacerations with fine chromic catgut. (Fig. 1.)

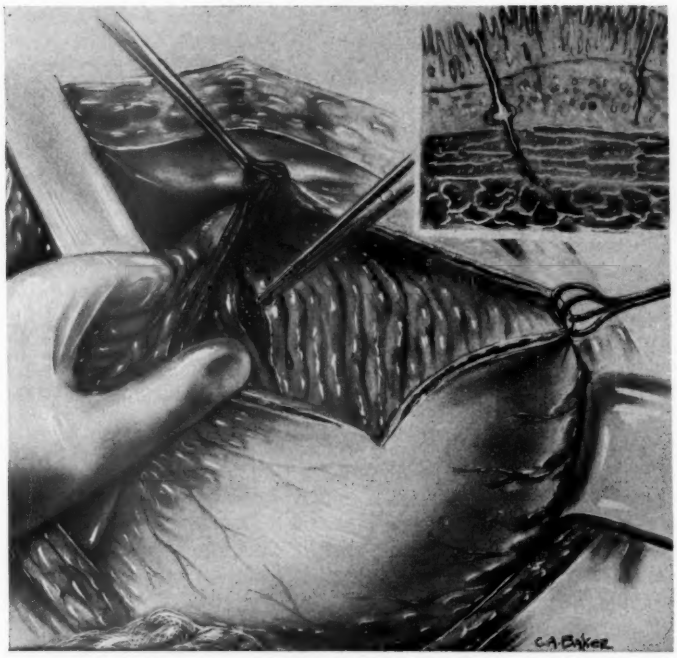

FIG. 1. Artist's illustration of findings in CASE 1. The large and small parallel tears are demonstrated with a clamp on the actively bleeding artery. Insert shows relative depth of the two lesions.

Postoperatively the patient did very well with the exception of a localized wound abscess which healed without incident. He has been instructed to make every effort to control bouts of vomiting in the future with large doses of antiemetic drugs.

CASE II. J. N., an eighty-three year old retired man, was admitted to the hospital on April 8, 1958, because of nausea and vomiting of coffee-ground material of several hours' duration. His past history was non-contributory except for the uneventful repair of a cervical esophageal diverticulum six years before.

On the afternoon of admission the patient had felt well until he visited his wife in the hospital where she had just undergone extensive surgery. He became emotionally upset and nauseated, and then vomited a considerable amount of normal gastric contents. Later the patient felt weak and nauseated again, and this time vomited a moderate amount of blood. He was then admitted to the hospital.

The initial hemoglobin was 11.2 gm. per cent, and it dropped to 10.4 gm. per cent by the next day. An initial roentgenogram demonstrated the possibility of a lesion on the greater curvature as well as a duodenal ulcer. A subsequent roentgenogram taken two days later was entirely negative and the deformities were assumed to be due to blood. During the next three days the patient received 7 units of blood which maintained his hemoglobin at about 9 gm. per cent. He was passing tarry stools daily. In view of the persistent oozing in a patient of this age, exploration was recommended and performed on April 12, 1958, with the diagnosis of gastric laceration as the prime probability.

This was confirmed at surgery, during which careful exploration revealed absolutely no disorder except two longitudinal lacerations near the cardia which were not bleeding at the time. Both were whipped over with continuous chromic catgut sutures and the procedure was terminated. His initial postoperative course was excellent. The hemoglobin rose to 12.6 gm. per cent after the administration of 2 more units of blood, and his preoperative blood urea nitrogen dropped from 65 to 35 mg./100 cc. However, he became extremely agitated and then manic on the afternoon of his second postoperative day. He required massive sedation for control and then grew steadily worse until his death the next morning. A postmortem examination confirmed the operative finding of no other disorder, and there was no evidence of further bleeding. The patient did have a bilateral broncho-

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pneumonia which was thought to be the cause of his death. At the time of death, his electrolytes were normal and temperature 100°F. at its highest. Unfortunately, permission for examination of the brain was not obtained.

COMMENTS

The description of the lesions in the original reports as well as in the subsequent cases, including the two just described, are all in close agreement. The lacerations occur in the longitudinal axis and are arranged in a radial manner anywhere about the cardioesophageal junction. They may appear in the esophagus or stomach alone, or extend into both. As already mentioned, they are frequently multiple. Up to five in a single case have been reported. The lacerations may extend completely through the muscularis mucosae. In nearly every instance the appearance of the laceration, either grossly or microscopically, is that of a fairly fresh tear although one or two cases have been reported with further complications of a chronic nature such as eventual delayed perforation. However, this syndrome should be distinctly separated clinically from spontaneous perforation of the esophagus which is a much better known and much more easily recognized entity.

There has been considerable speculation as to the exact etiology of these lacerations but no better explanation has ever been offered than the original concept of Mallory and Weiss [*1,2*] that the forces exerted at the time of vomiting cause these tears. It was their belief that the usual highly coordinated reflexes occurring in the act of vomiting became disturbed and the esophagus and diaphragm then fail to relax, particularly after repeated attacks of vomiting and retching. As a result of this, the gastric contents would be thrust with great force against the cardiac opening. In proving this theory they experimentally occluded the cardia of very freshly removed specimens, and by applying pressure to the distended stomach were able to reproduce exactly the types of laceration typically encountered in this syndrome. It is interesting that the vomiting which initiated the attacks of bleeding has been caused by many different precipitating factors including pernicious vomiting of pregnancy, motion sickness, postoperative nausea, uremia, acute gastrointestinal inflammation, excessive intake of alcohol and, in our own two cases, severe migraine headache and marked emotional upset.

Certain nutritional and other diseases might well contribute to the occurrence of these tears, as one would certainly anticipate a more fragile mucosa in the presence of severe nutritional imbalance or marked alcoholic or atrophic gastritis. However, despite the more frequent than average appearance of these factors in such cases, the one common denominator always present has been vomiting.

The interval from the time of vomiting to the time of definite proof of diagnosis has varied from a few hours up to twenty-one days. In the cases thus far reported, there has been a very decided preponderance of males but the total number to date is still insufficient to make this of any real statistical significance. The age incidence has also varied from twenty-one up to eighty-three years of age.

Of real significance as far as the literature is concerned is the fact that the seventeen verified cases reported in the three papers by Mallory and Weiss and later by Mallory and others were all diagnosed at postmortem examination, while all the lesions subsequent to that time have been identified either at the time of surgical exploration or endoscopic examination.

To increase the percentage of these cases recognized at postmortem examination, it would seem that there must be increased awareness by pathologists that such a lesion does exist. The very nature and appearance of the lesion makes it extremely easy to pass it off at the time of such a postmortem examination as due to the trauma of the autopsy procedure rather than to the actual cause of the massive bleeding. Furthermore, it has been suggested that the patients in this category are prone to fairly rapid exsanguination and therefore frequently become coroner's cases. The complete history and other important data are then often not available and the diagnosis is a little more readily missed.

To increase accuracy in diagnosing the syndrome clinically or surgically, the possibility of such a lesion being present in all cases of unexplained hemorrhage, especially massive upper gastrointestinal hemorrhage, must be kept in mind. If the history is clear enough with the story of vomiting normal stomach contents on one or more occasions prior to vomiting large amounts of blood or the passage of a large amount of blood, then one must be highly

suspicious of the presence of such a lesion even in the face of a history suggestive of ulcers or cirrhosis. In the absence of a history of an ulcer or cirrhosis, we were able to make a definite primary preoperative diagnosis of the Mallory-Weiss syndrome in our own two cases and confirm it by the finding of the typical lesions at the time of surgical exploration. Therefore, it would seem that history is by far the most important preoperative diagnostic sign. Roentgenograms, as would be anticipated, have been of no benefit whatsoever in diagnosing this lesion, and certainly clouded the issue in one of our patients. This has been further stressed in one of the reported cases which had already been diagnosed by gastroscopic examination and in which it was impossible to demonstrate the lesion by roentgenograms, although its exact location was known. The use of the gastroscope and esophagoscope in diagnosing the lesion was first mentioned by Mallory and Weiss [*1*] in their initial article in 1929, but no direct reference to this was made in any of their cases reported.

Hardy [*5*] (1956) was the first to give a definite description of the typical lesion as identified by the gastroscope. On the other hand, Kelley [*9*] reported a case in which he was unable to identify the lesion either by esophagoscope or gastroscope; about two hours following gastroscopy there was a recurrence of very massive hemorrhage requiring prompt surgical exploration. The typical lesions of the Mallory-Weiss syndrome were identified at surgery. While it was believed from the history that these had definitely been present prior to the introduction of the scopes, there was concern that the bleeding might have been reactivated by the attempted diagnostic procedure. In view of the hesitancy of many endoscopists to introduce the scope in the face of massive hemorrhage, the final diagnosis will probably depend on the findings at surgical exploration.

Treatment then would certainly start, as in most cases of extensive upper gastrointestinal hemorrhage, with at least a brief period of conservative management and careful observation.

The use of the Sengstaken tube has been tried by at least two other physicians and, as in one of my patients (Case 1), this failed to arrest the bleeding. It has been suggested that the pressure applied by the balloon is insufficient to control the arterial type of bleeding which is most frequently encountered with this syndrome. I would certainly agree with this suggestion.

When these measures have failed, as they did in the previously mentioned cases, prompt exploration is indicated. At the time of laparotomy, if there is no obvious cause of the bleeding such as a duodenal ulcer, malignancy or portal hypertension, it is recommended that a very generous anterior gastrotomy incision be made. Thorough inspection and palpation of all portions of the stomach and down into the duodenum is then possible. If the bleeding is very extensive at the time, making localization of the source rather difficult, it has been suggested that the mid-portion of the stomach be packed so that one can determine whether the bleeding is coming from above or below that level. With adequate retraction and good light, it should be possible to visualize the entire cardiac end of the stomach. In this manner, the great majority of the lesions will become readily visible. In those cases in which the bleeding is coming from above the cardia, it may be possible to palpate the lesion with the finger or attempt to visualize it through a sterile sigmoidoscope as was tried by Richards [*4*]. Once the lesion is visualized, a simple transfixion of any bleeding points with catgut sutures and closure of the laceration with the same type of material has been the treatment used by all of those reporting. Small and Ellis [*6*], and McPhedran [*8*], reporting lesions occurring in the esophagus, independently carried out identical treatment in which they closed the gastrotomy wound, completely mobilized the cardia and esophagus, and then repaired the laceration through an anterior opening in the esophagus. The results were good in both these cases.

The importance of recognizing and promptly handling the situation at surgery is very much in evidence in reviewing the cases in the literature, since the results of doing too little or too much at the time of exploration is so vital to the patient's welfare. There are cases reported in which the laceration was not identified at the time of exploration and since no other lesion could be found the patient was closed up without a diagnosis, only to exsanguinate subsequently. At postmortem examination the typical lacerations at the cardia with the bleeding vessel easily visible were found. On the other hand, total gastrectomy has also been performed when a similar patient continued to

bleed after a so-called blind subtotal gastrectomy for massive hematemesis.

CONCLUSIONS

The Mallory-Weiss syndrome, or massive bleeding from lacerations of the cardio-esophageal region subsequent to repeated vomiting or severe retching, while not commonplace, probably occurs much more frequently than one would judge from the reports in the literature. Two cases which seem entirely typical of this syndrome are presented, and a plea is made for increased awareness of this syndrome and proper management at the time of surgery.

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DISCUSSION

Victor Richards (San Francisco, Calif.): There are still many unanswered problems in the management of upper massive gastrointestinal bleeding. I have had no personal experience with this lesion. However, we have all been confronted with the problem of what to do with the patient with massive upper gastrointestinal bleeding after the initial decision as to whether or not he should be operated upon has been made.

There are many cases of bleeding which are never diagnosed, particularly in the aged.

Mallory and Weiss described the occurrence of this syndrome in the esophagus, and it is interesting that these cases have occurred primarily in the stomach. In theory, one would expect the lesion to occur in the stomach more frequently than in the esophagus because of the physical laws involved in rupturing an organ with a disruptive force against it.

Five years ago most of us would have thought that a blind gastrectomy should be performed when the source of gastrointestinal bleeding could not be found at surgery, and many surgeons still adhere to this principle. I agree with Dr. Etheredge that a long anterior gastrotomy should be performed rather than a routine blind gastrectomy.

One technical point which might be of help is to try to separate bleeding from below the esophagus from bleeding above the esophagogastric juncture. A Foley catheter is attached to the Levin tube, the balloon is inflated, and the catheter is pulled up to the esophagogastric juncture. This will effectively occlude the esophagogastric juncture and stop any blood from trickling down. One can then examine the upper end of the stomach carefully. This one point might be helpful in trying to localize some of these small tears in the gastric mucosa which might initiate, or be the source of, the bleeding.

Gradually, as the years go by, more and more of these problems can be solved. During the past year we have treated three patients (young and elderly) with gastrointestinal bleeding who surgically have been explored. Nothing was found. One of the patients underwent resection and bled again; another patient underwent gastrotomy which demonstrated no abnormalities, and then bled again. A blood dyscrasia is another possible causative factor which is more common, I think, than many of us realize. This is an absence of a specific blood-clotting factor. The lesion is described as pseudohemophilia, and the particular factor which is absent in the clotting mechanism is the Christmas factor.

People who are studying the details of the clotting mechanism have been able to demonstrate this as the causative factor in quite a few cases. Dr. Agler, who has been particularly interested in it, has reported a rather large series of patients who have an absence of this particular factor in the blood. I have treated three patients for recurrent massive bleeding during the past year who were surgically explored and nothing was found. Special studies of their blood demonstrated so-called pseudohemophilia, or absence of the Christmas factor.

This lesion is rather easily cured by giving the patients fresh blood which contains the Christmas factor. In stored blood it is not present in sufficient

concentration to stop the bleeding. In some of these patients with massive bleeding, fresh blood should be given before surgical intervention. Bleeding might cease by the administration of this specific factor in the clotting mechanism.

THOMAS FERGUSON: During the past year we have had the opportunity to operate on two patients in whom we have been able to demonstrate this lesion at the operating table. The second of these patients presented two features worthy of comment.

A thirty-four year old cirrhotic patient gave a history of ingesting large quantities of alcohol for the preceding ten years. His first symptom was that of massive hematemesis. Neither the patient nor his family, on close questioning before and after the diagnosis was proved, could recall any preceding vomiting, retching or gagging to account for the lesion.

It seemed logical to conclude that the laceration could occur without trauma or that the trauma could be so slight that the patient would be unable to recall the incident. A third possibility was that the anesthesia and amnesia which sometimes accompany the ingestion of alcohol might mask the event and prevent the eliciting of this history from the patient.

Gastric tamponade was employed in this particular patient since we were working with the incorrect diagnosis of bleeding gastric varices. It was effective in controlling bleeding for a forty-eight–hour period in this patient. Bleeding did recur, however, after release of tamponade, and the patient was subsequently taken to surgery for this reason.

Since the bleeding was controlled by the initial tamponade, it is possible that a second period of tamponade might have effectively controlled the bleeding and avoided the necessity of operation to control the bleeding.

CARLETON MATHEWSON, JR. (San Francisco, Calif.): Every physician who has encountered situations of massive gastrointestinal hemorrhage appreciates the difficulty that is encountered in exposing the esophagus and the cardiac end of the stomach from any sort of an incision in the stomach wall.

I agree with Dr. Etheredge that, when one is not able to find the source of hemorrhage, it is useful to open the stomach widely so that the cardiac end of the stomach, particularly, can be inspected. One will often find varices here which may be the source of hemorrhage.

A middle-aged Japanese woman, who had always been well, had had no previous gastrointestinal difficulty, and gave no history of alcoholism. She was awakened one night with violent retching and hematemesis, and entered the hospital very shortly thereafter. The patient had lost a tremendous amount of blood and was still having hematemesis. An attempt was made to restore her blood volume. However, she continued to vomit. She was taken to surgery and operated upon. The stomach was opened widely, and no source of hemorrhage was found.

It was noted at the time of surgery that she had some changes in her liver. This did not seem typical of cirrhosis, but in any event a biopsy of the liver was performed. The abdomen was closed and the patient was taken to the recovery room; within a very short period she started vomiting massively again. She was taken back to the operating room and the stomach was reopened. This time the esophagus was inspected and a laceration of the esophagus was found, and sutured.

She was then taken back to the ward, where she recovered and remained quite well. We thought that this was quite a typical case of the lesion that has just been described. She recently re-entered the hospital with a repeated episode of massive hemorrhage. On this occasion, we examined her more carefully because she had not lost quite as much blood. Very extensive esophageal varices were found. She was operated upon again and the source of bleeding was determined to have been from esophageal varices.

I have frequently seen lacerations of the wall of the stomach caused by manipulation in the attempts to find a source of bleeding that is not apparent when the abdomen is first opened.

SAMUEL N. ETHEREDGE (closing): I do not wish to leave the impression that this is a common lesion. We know it must be rare. However, it is one more source of bleeding that is worthy of consideration.

Another procedure has been repeatedly suggested, that is, packing the mid-portion of the stomach with a large laparotomy tape to ascertain which end of the stomach the bleeding is coming from. If the bleeding is coming from below the mid-portion, this procedure would be very helpful.

Much of the confusion about this syndrome originated from the initial reports of Mallory and Weiss. Many physicians believed that it was just another form of bleeding in cirrhotic patients with esophageal varices. Neither of our two patients had any history of ingesting large quantities of alcohol, and I think this is a particularly important point.

Reappraisal of the Surgical Treatment of Diverticulitis of the Sigmoid Colon

WITH SPECIAL REFERENCE TO THE CHOICE OF OPERATIVE PROCEDURE

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IN 1950 a twenty-year review of surgical treatment of diverticulitis of the colon at The Portland Clinic was reported [1]. Primary resection had been possible in four-fifths of all patients. In four-fifths of these, resection with anastomosis had been performed.

Furthermore, in patients requiring colostomy, prolonged diversion had not been necessary. The average duration of colostomy prior to resection had been four weeks, while total duration of colostomy had averaged nine weeks. This was in sharp contrast to the usual recommendation of fecal diversion for six to twelve months prior to definitive operation.

Smithwick (1942) [2] and Pemberton et al. (1947) [3] had pointed out that therapeutic measures short of ultimate resection resulted in prolonged morbidity, a high recurrence rate, and an unacceptable late mortality. Corroboration of these observations was evident in the study of patients in The Portland Clinic. Extension and liberalization of the indications for resection were recommended. Various plans of therapy, depending on the particular problem confronting the surgeon, were suggested.

To re-evaluate these recommendations, an appraisal of the results of treatment in the ensuing interval has been made.

MATERIAL AND CLINICAL PICTURE

The present review includes patients with diverticulitis of the sigmoid colon treated during the interval from January 1949 through December 1959—a period of eleven years immediately following the original study. Patients treated medically and those with associated carcinoma of the colon are not included. Forty-six patients had surgical treatment, and forty-four had resection in one or more stages.

In general, the clinical picture in the forty-six patients presented no striking change from that in the earlier study. For comparison, data have been compiled in Tables I and II to show age distribution as well as symptoms and signs. There were twenty-three men and twenty-three women.

Preoperative diagnoses are shown in Table III. The improvement in diagnosis reflected in this table is due, perhaps, to the fact that the previous study focused attention on the problems associated with diverticulitis. However, the well known problem of differentiating carcinoma of the sigmoid colon from diverticulitis is apparent. In four patients this differentiation was impossible prior to exploration.

INDICATIONS FOR OPERATION

Traditionally, operative treatment has been reserved only for patients suffering from the complications of diverticulitis (i.e., peritonitis, abscess, fistula, obstruction) or for those in whom the diagnosis of carcinoma could not be ruled out. Our previous review suggested broadening the indications for definitive resection in the belief that these complications could be avoided in many instances and the number of operations performed in stages could be reduced. The reports of Welch et al. [4], Judd and Mears [5], Lewis and Hurwitz [6], Stone and Garrett [7], Colcock [8], Greene [9], and others have since defined these indications more accurately.

Conclusions have gradually crystallized and are becoming more widely accepted. Definitive resection should be elected for patients who have recurring attacks of diverticulitis, urinary

TABLE I
AGE DISTRIBUTION

Age	Patients	
	No.	Per cent
31–37	2	4.3
40–49	9	19.5
50–59	16	35.0
60–69	12	26.0
70–79	6	13.0
84	1	2.2
Total	46	100.0

symptoms associated with an attack of diverticulitis, and severe persistent deformity demonstrated by roentgenogram after symptoms have subsided. Diverticulitis appearing in patients under fifty years of age is included by Welch et al. [4] as a cogent reason for advising elective resection.

Table IV lists the indications for resection in the present series. Most of the patients were operated on because of complications of diverticulitis. Eleven had resections at an interval between attacks. Failure of the condition to subside after several weeks of observation necessitated resection in two patients. One pa-

TABLE III
PREOPERATIVE DIAGNOSES

Preoperative Diagnoses	Patients	
	No.	Per cent
Diverticulitis	29	63.0
Carcinoma of colon	1	2.2
Questionable diverticulitis or carcinoma	3	6.5
Pelvic tumor	5	10.8
Pelvic abscess	1	2.2
Acute appendicitis	3	6.5
Acute abdomen	1	2.2
Perforated peptic ulcer	1	2.2
Small bowel obstruction	1	2.2
Questionable ruptured Meckel's diverticulum	1	2.2
Total	46	100.0

TABLE II
SIGNS AND SYMPTOMS (FORTY-SIX PATIENTS)

Signs or Symptoms	Patients	
	No.	Per cent
Inflammation*	30	65.2
Intestinal obstruction, complete or partial	2	4.3
Diarrhea	12	26.0
Rectal bleeding	8	17.4
Slight to moderate	7	
Severe	1	
Mass	13	28.2
Abdominal	4	
Pelvic	9	
Fistula	8	17.4
Sigmoidocutaneous	3	
Sigmoidovesical	2	
Sigmoidocervical	1	
Sigmoidoileocutaneous	2	

* Includes fever, chills, tenderness, rigidity, leukocytosis.

TABLE IV
INDICATIONS FOR RESECTION*

Indications	No. of Patients
Persistent attack	2
Recurrent attacks	11
Inability to rule out carcinoma	5
Perforated diverticulitis with peritonitis	9
Spreading	3
Generalized	6
Perforated diverticulitis with abscess	5
Intraperitoneal	2
Mesenteric	3
Obstruction	2
Fistula	8
Sigmoidocutaneous	3
Sigmoidovesical	2
Sigmoidocervical	1
Sigmoidoileocutaneous	2
Hemorrhage	1
Total	43

* Includes forty-two resections done at The Portland Clinic. Two resections done elsewhere (as first stage) have been deleted from this table. One patient, who died eight days after preliminary transverse colostomy has been included. One patient with sigmoid diverticulitis discovered at laparotomy had no surgical procedure performed on the colon.

tient required resection because of repeated massive hemorrhages.

OPERATIVE TREATMENT

Review of the recent literature reveals a variety of opinions as to the choice of surgical procedure under varying circumstances. It is generally agreed that in most instances elective resection at an interval between attacks can be accomplished safely in one stage. Various authors have reported resections in one stage in selected patients presenting the complications of fistula, small mesenteric abscess, or even mild obstruction. Our original report included a number of such cases. However, many authors still advocate preliminary transverse colostomy in almost all patients with the complications of fistula, obstruction, abscess, or peritonitis. As late as February 1960, Smithwick [*10*] urged such conservatism. This rule of thumb stresses fecal diversion for at least three months and, commonly, six months or longer prior to resection. Usually the Mikulicz or obstructive resection is rejected.

Table v shows the type of resection performed in the present series. Resection with anastomosis was elected twenty-six times out of forty-four, a complementary colostomy being added in three of the patients. Three-stage operations were employed in only eight instances. Mikulicz or obstructive resection was utilized in ten patients, or almost one-fourth of the total. When the cases in our two series are compared (Table v), it is apparent that one- and three-stage resections were equally frequent. Obstructive resection, on the other hand, was chosen almost twice as often in the present series.

This study, which brings thirty-one years of experience with diverticulitis up to date, has clarified, for us at least, various indications for selection of particular operations. We believe that there is a distinct place for a number of different procedures. We are convinced that there is no place for arbitrary and preconceived rules when considering an individual problem. To insist on preliminary colostomy for every complication of diverticulitis is to condemn many patients unnecessarily to prolonged morbidity and even increased risk. To fail to use colostomy under other circumstances may be equally lacking in judgment and result in devastating complications.

Primary Resection. Primary resection with anastomosis can be utilized with greater frequency than is commonly believed. With the widened indications for earlier operation in recurrent diverticulitis, elective resection and anastomosis after adequate preparation of the bowel is the procedure of choice in most instances. This applies as well to many patients whose disease cannot be differentiated from carcinoma of the colon. Small mesenteric abscesses may not contraindicate resection and anastomosis as in one patient in this series. However, severe grades of obstruction preclude this choice.

Certain complicated situations may easily adapt themselves to one-stage resections, as pointed out by Waugh and Walt [*11*]. Not uncommonly, a vesicosigmoidal or sigmoidocutaneous fistula may result from the involvement of a single diverticulum or of a short segment of inflamed colon without a surrounding abscess or wide zone of cellulitis. We have easily and safely resected five such lesions after careful preparation of the bowel, concluding the operations without colostomies. The length of time spent in the hospital was eight, ten, and eleven days for three patients. In two patients convalescence was more prolonged (twenty-four and thirty-five days) because of infection

TABLE V
RESECTIONS FOR SIGMOID DIVERTICULITIS

Operations	No. of Patients			
	1949–1959		1929–1948	
Resection with primary anastomosis		26		21
No colostomy or cecostomy	21		9	
Complementary colostomy	3		1	
Secondary colostomy	2		1	
Complementary cecostomy	0		10	
Obstructive resection		10		4
Resultant double-barreled colostomy	8		3	
End colostomy (distal limb closed)	2		1	
Three-stage resection		8		6
Transverse colostomy	8		6	
Resection and anastomosis	8		6	
Closure of colostomy	7*		6	
Total		44		31

* Third stage pending in one patient.

in the wound. There were no other significant complications.

Complementary Colostomy. Complementary transverse colostomy at the time of resection and anastomosis has a distinct place in surgical therapy for diverticulitis. We certainly cannot accept Smithwick's [*10*] insistence that it be done in every instance. We believe that anastomosis beneath the pelvic floor after resection of lesions located low in the sigmoid should be protected by colostomy. In certain patients the caliber of the colon at the site of anastomosis is small as a result of spasm. Colostomy may be indicated in these patients to permit healing before function is allowed. If the integrity of the suture line is in doubt for any other reason, colostomy will bring safety even though convalescence is prolonged. Waugh and Walt recommended utilization of this measure for similar indications.

We have no faith in cecostomy as protection for an anastomosis, and mention it simply to dismiss it. It has not been used in any patient since our original report.

Three-Stage Resection. Severe complications of diverticulitis often require colostomy before resection is attempted. We believe, however, that discriminating judgment is of great importance in determining the need for preliminary diversion. Obviously, it is indicated in partial or complete obstruction as in one patient in this series. Occasionally minor degrees of obstruction may be relieved by conservative management, permitting elective resection, as in one patient in this series. If a retroperitoneal or localized intraperitoneal abscess results from perforation, the bowel should be put at rest at the time of drainage of the abscess as in two patients in this series. Fistulas (cutaneous, vesical, or vaginal) with wide cellulitis or severe fixation should not be attacked prior to colostomy diversion. In three such patients in this series, preliminary colostomy was performed. However, we are convinced that the use of this method for all or most of the complications of diverticulitis is not only unnecessary but unwise. This practice has resulted from acceptance of arbitrary rules which should be discarded.

Colostomy and Colostomy Closure. Colostomies for fecal diversion should always be placed in the transverse colon. Future resection may be impeded unless this rule is followed.

We still agree with the conclusions in our original report [*1*] relative to the duration of fecal diversion. Only rarely should it be necessary to delay resection six to nine months.

As pointed out by Mayo and Blunt [*12*], it is fallacious to expect the severe cystitis incident to vesicosigmoidal fistula to clear after fecal diversion. Urinary infection will always persist until the fistula is separated. Surrounding inflammation and cellulitis should recede enough a few weeks after colostomy to permit resection with safety and without the risk of prolonged urinary sepsis. In our series preliminary colostomy for this complication was not necessary.

When preliminary colostomy has been elected for perforation with peritonitis, abscess, or cutaneous fecal fistula, each situation must be assessed on its own merits. As in the case of interval appendectomy after drainage of an appendiceal abscess, definitive operation can usually be done in from six weeks to three months. In the exceptional problem greater delay may be necessary.

In our series eight patients required three-stage operations. In one patient resection was delayed eight and one-half months following colostomy. Definitive resection was promptly performed in one patient who came to us six months after colostomy had been performed elsewhere. In the remaining six patients the duration of colostomy averaged two and one-half months, varying from seven to fourteen weeks.

We do not agree with Welch et al. [*4*] that a totally diverting colostomy, utilizing a skin bridge between the limbs, is necessary. On the contrary, we perform a loop colostomy, preferring to make the opening in the long axis of the bowel to simplify closure. The small amounts of material escaping diversion are inconsequential. Closure of such a colostomy is a relatively minor procedure compared to end-to-end anastomosis. There has been no instance of leak at the site of closure. Hospitalization has averaged just over one week.

Prior to closure we always check the anastomosis by barium study. This has been rewarding occasionally in pointing up minor leaks or demonstrating constriction from edema or induration at the anastomosis. Such complications have always yielded to time.

Obstructive Resection. Pemberton et al. [*3*] (1947) reported the use of obstructive resection, with or without preliminary colostomy, in

FIG. 1. Plain film of abdomen showing massive amount of fecal material throughout the colon. The patient was an eighty-four year old woman who had generalized peritonitis from a large perforation of the sigmoid in the area of severe diverticulitis. Thick liquid stool was pouring from the perforation at laparotomy. Recovery followed obstructive resection.

three-fourths of their patients treated between 1941 and 1945. However, most authors have neither chosen this method nor approved its use. In 1953, Welch et al. [*4*] had discarded it "except in the rare instance in which a free perforation can be exteriorized easily."

Our original study included four such cases. Since then, we have acquired a greater appreciation of the value of this procedure in selected cases of complicated diverticulitis. We have been encouraged to continue its application by the reports of Mayo and Blunt [*12,13*] and of Gilchrist and Economou [*14*] despite the disparagement of this method by others.

We have used two variations of this operation in ten patients. Resection with elevation of both limbs into the wound as a double-barreled colostomy was used in eight patients, and resection with closure of the distal limb and end-colostomy of the proximal limb was used in two patients. Bowel continuity is re-established at a second-stage operation.

What are the indications for obstructive resection and why do we favor it over the three-stage resection? Its most frequent use in our hands, as in the series of Gilchrist and Economou [*14*], has been for free perforation of diverticulitis with general or spreading peritonitis, or perforations with early localized peritonitis before a definite abscess has developed. In a few such patients the involved segment of colon with the site of perforation is so situated that it can be resected and the two ends easily exteriorized. However, this is often impossible without freeing the lateral reflection and, occasionally, even the medial reflection of the peritoneum. We have not hesitated to utilize these simple maneuvers. When the perforation or surrounding involvement is in the low sigmoid, or rarely in the upper rectum and the distal limb cannot be exteriorized, it may be closed and only the proximal end elevated into the wound. It has always been possible to resect well beyond the zone of inflammation. Thus, the site of perforation, at times so difficult or impossible to close securely, is eliminated expeditiously and continuing leak or recurrent perforation is prevented.

We are convinced that this may be a life-saving procedure. Election of transverse colostomy for free perforation with peritonitis disregards the fact that considerable fecal material often remains in the segment between the colostomy and the perforation. (Fig. 1.) Continued leakage of this material may result in overwhelming contamination despite every effort at adequate drainage. We experienced one such result in this series (Case 4, Table VIII).

It is well known that patients with intestinal perforations from other causes seldom succumb if the source of the contamination is eliminated early. One would quail at the suggestion of proximal diversion as a method of treatment for perforations of the appendix or traumatic perforation of the small bowel. Results of treatment of large bowel perforations in World War II are well known. Exteriorization was undoubtedly the most important factor in reducing mortality from such injuries. The application of these same principles for free perforations complicating diverticulitis would seem logical. In our present series nine patients with acute perforations and peritonitis of varying degrees underwent obstructive resection without mortality.

The same principle can be applied in patients with fistula, as reported by Mayo and Blunt [*12*]. In our series one patient with a fistula between the sigmoid and the cervical stump was managed nicely by obstructive resection.

Extent of Resection. No matter what method of resection is elected, wide removal of the involved segment is required. However, a generous margin at each end of the inflamed

TABLE VI
COMPARISON OF MORBIDITY

Operations	No. of Patients	Averages		
		No. of Days Hospitalized	No. of Weeks of Disability*	No. of Weeks Before Colostomy Closure
Resection without colostomy	20	14.8	2.1	
Obstructive resection	9	30.6	12.3	10.9
Resultant double-barreled colostomy	7†			
End colostomy (distal limb closed)	2			
Resection with proximal colostomy	10	33.9	15.2	14.0
Colostomy as first stage	6‡			
Complementary colostomy	3			
Subsequent colostomy	1			

* From first hospital admission until final discharge. Many patients had multiple hospital admissions.

† One patient delayed colostomy closure for three years for personal reasons and has been deleted from this table.

‡ One patient awaiting colostomy closure has been deleted from this table. All deaths have been eliminated from this table.

colon, to assure safe anastomosis, is sufficient. Recurrences of diverticulitis characteristically occur in the segment previously involved. We have had no recurrence in our series despite the frequent presence of diverticula adjacent to the suture line. Review of the literature indicates that diverticulitis after such resections must be rare indeed.

Removal of all of the sigmoid necessitates mobilization of the descending colon and the splenic angle in order to permit anastomosis. If undertaken routinely, as suggested by Smithwick [*10*], this maneuver would certainly increase the operative risk.

MORBIDITY AND MORTALITY

In an effort to show differences in morbidity following various types of resection, we have tabulated average days of hospitalization, average duration of disability from the first admission to final hospital discharge, and average duration of colostomy (Table VI).

Hospital stay of patients requiring colostomy was almost twice that of patients undergoing one-stage resection. Duration of disability averaged six to seven times longer than in patients who did not require fecal diversion. When compared to obstructive resection, three-stage resection increased the average total disability by three weeks and the duration of colostomy by one month. These facts should suggest to thoughtful surgeons a careful review of their own indications for selection of the various approaches to definitive operation in diverticulitis of the sigmoid colon.

TABLE VII
THIRTY POSTOPERATIVE COMPLICATIONS IN TWENTY-ONE PATIENTS

Complications	No. of Patients	No. of Deaths
Pneumonia	2	
Thrombophlebitis	1	
Coronary thrombosis	1	1
Cardiac decompensation	1	
Wound dehiscence	3	
Wound infection	11	
Paralytic ileus	2	
Peritonitis		
Leak sigmoid anastomosis	2	1
Cecal perforation after colostomy closure (third stage)	1	1
Small bowel obstruction	3	
Severe diarrhea	1	
Oliguria and electrolyte imbalance	1	
Toxic psychosis	1	

Table VII lists the postoperative complications. Of the total forty-six patients in our series, twenty-one had thirty complications. These complications were responsible for three of the four deaths in the series.

TABLE VIII
DEATHS

Case	Age, Sex	Cause
1	46, M	Coronary thrombosis fourteen days after primary resection with anastomosis
2	67, F	Generalized peritonitis twenty-five days after sigmoid colectomy and primary anastomosis due to a leak at anastomosis. Transverse colostomy five days after resection.
3	62, M	Generalized peritonitis six days after closure of transverse colostomy (final stage of three-stage procedure). Cecal perforation occurred due to obstruction of distal colon due to unrecognized left subphrenic abscess.
4	64, M	Generalized peritonitis eight days after transverse colostomy and pelvic drainage for sigmoid diverticulitis with perforation.

The pertinent details in the course of each of the patients who died are summarized in Table VIII. Two of the deaths (Cases 2 and 3) might well have been avoided and should not necessarily be attributed to the method of treatment elected.

In Case 2 the traumatized edges of bowel, released from a crushing clamp, were inadvertently utilized in the anastomosis at the time of resection for what was thought to be carcinoma of the sigmoid. When breakdown occurred, ten hours had elapsed after obvious anastomotic leakage before the surgeon was notified of any change in the patient's symptoms or condition. She succumbed to generalized peritonitis despite local drainage and proximal colostomy. Elevation of the anastomosis into the wound instead of transverse colostomy might have prevented this fatality. Complementary colostomy at the time of the resection undoubtedly would have prevented this death.

One patient (Case 3) presented a knotty problem. The patient was a sixty-two year old man with a free perforation of the sigmoid and generalized peritonitis. Transverse colostomy and local drainage were immediately carried out. Pelvic abscesses were drained transrectally on two occasions a few weeks after the original peritonitis. Resection was accomplished without major incident after maintenance of fecal diversion for eight and one-half months. This was the only patient in our series with colostomy established for more than three and one-half months prior to resection. Retrograde barium study of the anastomosis, the descending and the transverse colon showed no obstruction. Closure of the colostomy was performed five weeks after resection in the presence of an unsuspected left subphrenic abscess. The abscess, which was detected at autopsy, had produced complete obstruction of the splenic angle of the colon. Clinically the situation had been interpreted as paralytic ileus. The obstruction resulted in perforation of the cecum with lethal peritonitis despite cecostomy and exteriorization of the recently closed transverse loop of the colostomy. In retrospect, one wonders if the selection of obstructive resection as the original procedure, to avoid continuing contamination at the site of perforation, might not have effected a different end result.

The fourth death was due to peritonitis from free perforation of the sigmoid despite transverse colostomy and local drainage. Here, too, obstructive resection might have altered the course.

SUMMARY

The surgical treatment of diverticulitis of the sigmoid colon at The Portland Clinic during the interval from January 1949 through December 1959 has been reviewed. Resection of the sigmoid was performed in forty-four out of a total of forty-six patients.

Primary resection was accomplished in thirty-six patients (80 per cent). Ten of these had obstructive resections. Three-stage procedures were elected in only eight patients. This series is compared to a series in the preceding twenty years and previously reported.

Morbidity has been greatly reduced not only by the frequent selection of primary resection but also by reduction in the duration of colostomy prior to resection in procedures performed in stages. Critical evaluation of the four deaths in the series reveals that this lowered morbidity has been accomplished without increasing the surgical risk incident to treatment of diverticulitis and its complications.

The several indications for election of various methods of treatment have been considered in detail. It is of great importance to consider each situation individually rather than to accept the arbitrary rules of therapy suggested in the past. Many patients with recurring attacks of diverticulitis should undergo definitive operation at an elective interval to avoid the severe complications of peritonitis, abscess, obstruction, and fistula. Study of the present series indicates that resection and anastomosis can be completed safely in many situations heretofore thought to require three-stage procedures.

Obstructive resection has proved to be highly effective, particularly for free perforation with peritonitis. In such situations we prefer this method to the alternative of drainage and proximal colostomy since its use prevents continuing contamination and permits immediate elimination of the focus of infection. The dictum that obstructive resection is usually impossible or unwise has been shown to be mistaken. The disfavor into which this method has fallen is undeserved.

Thoughtful individualization of each problem resulting from diverticulitis of the sigmoid colon will permit early elimination of the offending segment of bowel with safety in the great

majority of patients. Only in this way can morbidity be reduced.

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DISCUSSION

Caleb Stone (Seattle, Wash.): Dr. Boyden emphasized the importance and feasibility of early one-stage resection and anastomosis in the treatment of the chronic and many other forms of acute diverticulitis in 1949. His present paper, reviewing the experience of the last ten years, confirms the wisdom of his earlier recommendations.

Our experience with diverticulitis, and our concept of the treatment of it, closely parallels that of Dr. Boyden. In our series, 75 per cent of the patients were treated by a one-stage resection with primary anastomosis, 17 per cent were managed by a two-stage procedure and 8 per cent by a three-stage procedure. Morbidity has been under 2 per cent.

I would add a fifth indication for early elective operation to the four Dr. Boyden mentioned—namely, bleeding, either mild or severe, originating in the colon, which is the site of localized diverticulosis or diverticulitis as indicated by roentgenographic examinations.

I would also modify his statement that persistent, severe deformity of the colon on roentgenographic examination, following an attack or attacks of diverticulitis, justifies resection. We would prefer to omit the qualifying adjective "severe" because a degree of inflammatory change is often present in the colon which was not measured by the roentgenographic study. A polyp or an early cancer associated with diverticulosis, and unrecognized on roentgenograms, has produced a moderate persistent deformity.

The effectiveness of obstructive resection in the treatment of acute diverticulitis, particularly in cases of free perforation with peritonitis, has been stressed. He has included in this category, not only the classical obstructive resection described by Dr. Rankin, but also various modifications of it. When the involved segment of the sigmoid can be entirely exteriorized and for various reasons primary anastomosis is not practical, the classical obstructive resection is ideal. Under these conditions it permits the removal of the acutely involved bowel, and definitive treatment can be completed in a second procedure.

Often, the acute inflammatory process is too low in the sigmoid to permit resection and the formation of a double-barreled colostomy. We have elected to resect the damaged bowel in several such situations, closing the distal stump and bringing the proximal limb of the colon out as a single-barreled functioning colostomy. This procedure has been particularly useful in instances in which the distal segment of bowel has been too friable to permit a safe anastomosis, or it has been unwise at the time of resection to attempt mobilization of the left colon and splenic flexure to obtain sufficient length of bowel to accomplish anastomosis because of local peritonitis, age of the patient, or obesity.

When resection can be safely accomplished in one or the other of these ways, it is preferable to the drainage of an abscess about the sigmoid and proximal transverse colostomy, because it not only removes the diseased bowel, but prevents further contamination from the column of feces distal to the colostomy.

In the closure of the double-barreled colostomy, or the re-establishment of bowel continuity after resection and single-barreled colostomy, the importance of resecting the entire sigmoid and creating anastomosis with the rectum should be emphasized. The sigmoid colon is most frequently the site of both diverticulosis and diverticulitis, and diverticula left in the distal sigmoid may cause

trouble later. Therefore, I would caution against attempting re-anastomosis too soon after removal of the inflammatory bowel, lest unresolved inflammatory reaction in the distal segment produce poor healing, fibrosis, and narrowing of the anastomotic stoma.

I believe it is helpful to utilize the end-to-side anastomosis described by Dr. Baker some years ago when doing a low anastomosis with the rectum. This procedure, when done at a low level, is easier to perform than an end-to-end anastomosis and it also offers better assurance of a wide anastomotic ring.

Acute diverticulitis and its complications present a real challenge. Certainly, seasoned judgment on the part of the surgeon, and a wide variety of surgical procedures to select from, are necessary in the satisfactory management of this condition. Dr. Boyden has pointed out many of the pitfalls that may confront the surgeon and has indicated the reward of a bold, but carefully planned, surgical procedure.

Grosvenor T. Root (Oakland, Calif.): One may frequently remove the inflammatory lesions involving the left colon when they are first encountered by performing obstructive resection or primary anastomosis without, in many instances, a proximal colostomy. However, Dr. Boyden did have to perform a transverse colostomy in eight out of the forty-three patients, because it was not possible to resect the colon initially in some instances.

At the subsequent operation, when it is possible to resect the left colon, the same aggressive attitude should be maintained, and at that time one can resect the transverse colostomy if the anastomosis in the left colon is satisfactory. This procedure has many advantages. One obviously avoids any further operative procedure with the accompanying problems, and the improvement in the mental attitude of the patient in the older age group is amazing when he or she is informed that the lesion in the left colon has been resected and the colostomy no longer exists. Patients tolerate a permanent colostomy extremely well. However, such tolerance is woefully lacking in a patient who has a temporary colostomy. Also, the danger of infecting the operative site is reduced as the colostomy is no longer present.

Finally, the procedure is much simpler from a technical standpoint when the abdomen is open. It is a much quicker and easier matter to excise the colostomy, anastomose the two ends of the bowel and repair the ventral defect.

The necessity of performing a proximal colostomy is decreasing. However, Dr. Burt Christensen (my associate) and I were very pleased with the results when we were able to excise the transverse colostomy at the same time that we resected the lesion involving the left colon in five of the last seven patients on whom it was necessary for us to perform a transverse colostomy. In two instances we did not believe that it would be safe to resect the colostomy at the time, and were very pleased to have it present as a safety measure.

Siegfried F. Herrmann (Tacoma, Wash.): I am very sorry that Dr. Boyden did not have time to develop his idea of advocating obstructive resection for diverticulitis, because I cannot understand that advocacy.

Perhaps I was one of the first surgeons on the Pacific Coast to make a plea for surgery for diverticulitis before this Society in 1948. At that time, I pointed out the very serious complications that result from conservative management. I believed that we should regard this disease as a surgical problem because of these complications.

Among those cases which I presented to this Society were two in which I had thought I made mistakes. They were both cases of obstructive resections. One of these patients had an acute perforation. I had thought the patient had appendicitis, and found that it was an acute perforation of diverticulitis. The colon was exteriorized as in an obstructive resection, and the patient got a severe infection in the abdominal wall, later had a hernia, and required more surgery. A primary anastomosis may have been the operation of choice.

Another patient had an obstructive resection when there was chronic diverticulitis with much inflammatory change in the mesosigmoid. I thought I could exteriorize this, remove the affected portion and do an obstructive resection. This patient died of a pulmonary embolus. She developed an iliac thrombosis because this obstructive resection was done right over the iliac vessels. The infection spread to a vein. Resection should have been deferred.

I ceased performing obstructive resection for diverticulitis or carcinoma. If the colon can be mobilized, a primary anastomosis is usually performed for diverticulitis.

I pointed out previously that it is not easy to make a diagnosis, and that one might easily think the diagnosis is diverticulitis when it actually is cancer. A patient who was a nurse came to me in March 1958. She complained that she had pain in her left lower quadrant when she moved or strained. She said it had started during the previous December while she was reaching for something. The patient was quite obese. I felt a little tender spot which I thought was a lump of fat protruding through the fascia.

She returned three months later with a temperature of 102°F., severe chills, and very marked abdominal discomfort. Marked tenderness was present along the course of the sigmoid and descending colon. I believed that this was caused by

diverticulitis, that perforation had occurred, and that an abscess had developed. I thought a roentgenographic examination should not be performed at the time, so I simply treated her conservatively and gave her antibiotics. The abscess, I thought, might absorb. However, it did not absorb. The abscess was drained, and the sigmoid was later resected for what was thought to be diverticulitis, but it was a cancer. The woman died several months later of her carcinoma. The clinical picture was one of acute diverticulitis, but it was a surgical disease.

LEON GOLDMAN (San Francisco, Calif.): Diverticulitis seems to be increasing in incidence. Its complications may be prolonged and subject the patient to a long period of invalidism. Free perforations of the colon may require some type of exteriorization procedure.

The indications for surgery are such that we elect to operate on patients with diverticulitis more frequently than we did during the previous decade. One of the indications for operation mentioned by Dr. Boyden was the inability to rule out carcinoma, particularly in those lesions in the sigmoid colon.

We recently reviewed our experiences with this particular problem, thinking that as a result of such a study we would perhaps be able to diagnose carcinoma of the sigmoid colon more accurately, particularly when a problem of differential diagnosis between diverticulitis and carcinoma arose. We reviewed a group of 199 patients who had carcinoma alone, diverticulitis alone, or a combination of the two.

We were surprised to find that 20 per cent of the patients with diverticulitis of the sigmoid colon had an associated benign or malignant neoplasm. Approximately 10 per cent had a malignant type of polyp. This incidence of malignant polyps has led us to operate upon patients with recurrent or chronic diverticulitis sooner.

The clinical findings in all the patients were reviewed to see if some sort of pattern would emerge. Many of the symptoms were of no value in making a differential diagnosis. Only 3 per cent of patients with carcinoma alone had had a previous attack, but 53 per cent of patients with diverticulitis alone had had a previous attack. Eighty-two per cent of patients with carcinoma of the sigmoid colon had a history of bleeding or evidence of it upon examination while 37 per cent of patients with diverticulitis had such a history. Anemia, as exemplified by a hemoglobin value below 12 gm. per cent, was present in 40 per cent of the patients with malignancy and 33 per cent of those with diverticulitis. Seventy-three per cent of patients with carcinoma of the sigmoid and 28 per cent of patients with diverticulitis had a history of weight loss before the present attack.

After reviewing the history and clinical findings we found that there was no distinguishing pathognomonic feature in a given patient that could help us to make a differential diagnosis.

We reviewed the diagnostic accuracy of barium enema and found that when carcinoma existed alone, or diverticulitis existed alone, the radiologist's diagnosis was accurate approximately 94 per cent of the time. When carcinoma and diverticula or diverticulitis were present simultaneously, the incidence of a correct diagnosis was only 69 per cent.

We even found errors in differential diagnosis at the operating table. In those patients who had carcinoma without diverticula, the incidence of a correct diagnosis was 98 per cent. In those patients who had both carcinoma and diverticulitis a correct diagnosis was made at the operating table in only 79 per cent; and in patients who had diverticulitis alone, a correct diagnosis was made by the surgeon in only 72 per cent. The surgeon was in error in 21 per cent of all cases even when the lesion was seen at operation. I think we all agree that a different type of operative procedure should be performed for known malignancy as compared to known diverticulitis.

It is very difficult to make a differential diagnosis in patients who have rows of diverticula in the colon with a minor defect which may be due to an early polyp or carcinomatous lesion. Here early surgery is indicated.

ALLEN M. BOYDEN (closing): I am grateful for all of the comments that have been made by all of the discussants. I will discuss a few of these separately.

I agree with Dr. Stone about the use of the word "severe." Any real continuing deformity other than just the presence of diverticula would give us concern.

Time did not permit a discussion of the extent of resection for diverticulitis. I cannot agree entirely with Dr. Stone about this. The vast majority of recurring attacks will be found to occur in the same area or in the same group of diverticula that were involved originally. I think this is borne out in any study of the literature. We can see no reason for resecting a great amount of colon which is not involved in inflammation.

If one resects all of the sigmoid, by definition this means that every patient, many of them elderly, will have to have extensive resection with mobilization of the splenic flexure in order to bring the descending colon to the rectum. Dr. Smithwick suggested this in his last report, and I disagree with him. We know of no recurrence of diverticulitis in the sigmoid when a generous margin of uninflamed bowel is removed. The anastomosis can be made immediately through areas where diverticula are present, beyond the zone of inflammation. Cer-

tainly when diverticula involve the descendens and the transversus, this would also apply.

We have not closed the transverse colostomy at the time of sigmoid resection, as suggested by Dr. Root. When we make a preliminary colostomy we are happy to have its protection after the second-stage resection. Actually we do not believe in "resecting" the colostomy, or in completely diverting colostomies, as suggested by Welch. We bring out a loop, opening it longitudinally. It is closed transversely after the anastomosis has healed. Closure is a simple procedure, requiring a week or less of hospitalization. However, I am sure there must be situations in which this third stage can be safely avoided by following Dr. Root's suggestion.

I might remind Dr. Herrmann that one of his preceptors, or at least one of his associates in the past at the Mayo Clinic, Dr. Pemberton, reported that three-quarters of their total cases were treated by this method up to 1945. I believe we stand in good company to recommend obstructive resection. The paper will express the reasons for electing this operation.

I am happy that Dr. Goldman elaborated on the association of carcinoma and diverticulitis, or rather the fact that a carcinoma may develop in people with diverticula and diverticulitis. Obviously there is no association in etiology here. Patients with coexistent carcinoma of the colon were not included in our study.

The Management of Bleeding Diverticula of the Colon

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IT has been stated by surgeons of great experience that diverticula of the colon rarely, if ever, hemorrhage and that bleeding, even in small amounts, seldom occurs. There are other surgeons of equal experience who find that bleeding commonly occurs from both diverticulosis and diverticulitis. These two conditions are often spoken of as diverticular disease. This controversy has been going on for a number of years. It has been of sufficient intensity to cause me to become very much interested in our cases of diverticular disease at Good Samaritan Hospital.

Heinemann [1] of Good Samaritan Hospital Pathological Laboratory recently stated that bleeding points in diverticular disease of the colon could almost never be found, even at autopsy, although it was certain that the bleeding occurred from the diverticular disease of the colon and from no other lesion in the bowel.

A solitary diverticulum of the colon rarely occurs. Nevertheless, it has been said that solitary diverticula bleed more often than multiple diverticula occurring in the colon. Young [2] reports a case of a single inverted diverticulum of the transverse colon with massive bleeding. Maynard [3] also reports one case of massive bleeding from an ulcerated base of the diverticulum in which there were several small arteries immediately beneath the ulceration. Sections through other inverted diverticula disclosed the presence of large vessels near the mucosal surface with evidence of inflammation. Inverted diverticula are not common in our experience.

Their impression is that an inverted diverticulum has the same tendency to bleed as a polyp. Fairly large vessels at the base of the diverticulum are often adherent to it. When the diverticulum inverts, these vessels are carried with it providing a rich source for massive hemorrhage if erosion of the mucosa occurs. Bleeding in diverticulitis has often been attributed to concomitant polyposis. It is difficult to differentiate grossly between an inverted diverticulum and a polyp. Graser and Edwards [4] thought the diverticula occurred along the source of a perforating vessel.

The purpose of this paper is to discuss the various methods of diagnosis and treatment used in bleeding from diverticular disease. It is not submitted as an attempt to produce any controversy as to the type of treatment which should be employed. It is a study of our cases of diverticular disease of the colon with special reference to bleeding.

INCIDENCE

On the surgical services at Good Samaritan Hospital a total of 250 patients with diverticular disease were treated during the past ten years (1949 to 1959). In fifty of these, some bleeding occurred and diverticular disease was thought to be the sole cause.

However, there were actually only twenty cases in which the bleeding was heavy enough to warrant giving the patient at least one transfusion, and in one case sixteen transfusions were required. Eight per cent of 250 patients required one transfusion or more. Only one death occurred in this series. There were seven other cases in which bleeding was thought to occur along with other pathology. In these seven cases the primary cause was not diverticular disease. There were two cases of carcinoma of the sigmoid, two of carcinoma of the rectum, two of small hiatal hernias and one of a polyp with questionable malignant degeneration. Eighteen of the fifty cases were listed as diverticulitis and thirty-two as diverticulosis.

Table 1 shows the incidence of bleeding in the Good Samaritan Hospital series.

TABLE I
SYMPTOMS OF DIVERTICULOSIS AND DIVERTICULITIS AND THEIR TREATMENT

Symptoms and Treatment		No. of Patients
Hemorrhage		50
Operation	7	
No operation	43	
Other Symptoms		200
Resection	26	
Other operations	22	
No operations	152	
Total		250

TABLE II
AGE DISTRIBUTION—PATIENTS WITH BLEEDING FROM DIVERTICULAR DISEASE

Age	No. of Patients
40–49	1
50–59	9
60–69	13
70–79	15
80–89	9
90–99	3
Total	50

The incidence of diverticulosis of the colon has been recognized to be from 5 to 10 per cent in various series. Sauer [5] found the incidence 5.2 per cent in 70,572 patients. Pemberton [6] found it to be 8.5 per cent. Ransom [7] found that one in every eight among 1,764 cases had either diverticulitis, diverticulosis or diverticulitis with complications. In 1927 deQuervain [8] discussed diverticulitis and described a hemorrhagic form which often was accompanied by sudden severe bleeding.

Rankin and Brown [9] believe that 15 to 20 per cent of people with diverticulosis will develop diverticulitis. Neal [10], Noer [11] and many others believe that diverticula of the colon cause hemorrhage and that it occurs quite frequently. Stone [12] has pointed out that the most important symptom of diverticulitis may be sudden, profuse hemorrhage.

In Ransom's [7] series, colonic hemorrhage occurred in nine out of fifty-three (17 per cent) cases of diverticula of the colon.

Noer [11] gives the incidence of bleeding from various series as follows: there were 324 of 2,896 cases which showed rectal bleeding; the lowest percentage was 3 per cent and the highest 47 per cent in this series; the next highest was 41 per cent from Henry Ford Hospital and reports from Baylor Hospital stated 30 per cent.

TABLE III
DURATION OF BLEEDING—PATIENTS WITH DIVERTICULAR DISEASE

Duration of Bleeding	No. of Patients
1 to 7 days	29
1 wk to 1 mo.	6
1 mo. to 6 mo.	9
6 mo. to 1 yr.	6
Total	50

ANALYSIS OF CASES

The patients ranged in age from forty to ninety-nine years and the average was seventy-four years. This is to be expected as it is a disease of the older age group. Diverticular disease, in our experience and that of others, is rare before the age of forty years (Table II).

DURATION OF BLEEDING

The duration of the hemorrhage ranged from one day to one year. We believe that those patients who bled over a period of one month to one year had diverticulitis (Table III).

SYMPTOMS AND PHYSICAL SIGNS

Nausea, vomiting and diarrhea often accompanied by abdominal distress along with

TABLE IV
SIGNS AND SYMPTOMS—PATIENTS WITH BLEEDING FROM DIVERTICULAR DISEASE (FIFTY CASES)

Symptoms and Signs	No. of Patients
Symptoms:	
Colonic bleeding	50
Diarrhea	19
Abdominal distress	25
Nausea and vomiting	13
Constipation	20
Physical signs:	
General abdominal tenderness	13
Tenderness in the lower quadrants	10
Mass	3
None	24

TABLE V
SITES OF BLEEDING IN DIVERTICULAR DISEASE

Site	No. of Cases
Cecum	2
Ascending colon	8
Transverse colon	9
Descending colon	22
Rectosigmoid area	48

colonic bleeding were the usual symptoms (Table IV). Sudden severe bleeding occurred in ten cases. A high percentage of these patients gave a past history indicative of spastic and irritable colon.

In this series of fifty patients who had bleeding diverticular disease, tenderness over the lower quadrant was noted frequently. Pallor and air hunger was noted in a few patients, especially those who had a considerable loss of blood and required from four to sixteen transfusions.

The various areas in which diverticular disease usually occurs are shown in Table V. It is almost impossible to definitely pinpoint bleeding diverticula in all cases, Circumstantial evidence was the only evidence in some cases in which bleeding occurred from diverticular disease.

DIAGNOSIS

Quinn and Oschner [*13*] have set out the following criteria to be fulfilled before bleeding from the colon may be attributed to diverticular disease. Keith and Rini [*14*], as well as others, have accepted this in making the diagnosis of bleeding from the diverticula.

1. Passage by rectum of either bright or dark red blood.
2. Use of barium enema, sigmoidoscopy and contrast air studies, in showing diverticular disease, all other bleeding lesions being excluded as nearly as possible.
3. Roentgenograms of the stomach and small intestine showing no abnormalities.
4. Normal blood coagubility and blood picture.

Roentgenographic evidence of diverticular disease in the absence of other pathology was the usual method in which the diagnosis of diverticular disease was made. Blood found by sigmoidoscopic examination at surgery or by microscopic sections coming directly from the submucosal vessels was excellent evidence that the bleeding came from the diverticulum. Franco [*15*] found severe bleeding in ten of forty-six cases of diverticular disease of the colon. However, Scarborough and Klein [*15*] reported that they removed polypoid tumors from sixteen patients in whom bleeding had been thought to occur from diverticular disease. They have more recently reported on additional patients in whom malignant or polypoid tumors were removed and in whom the bleeding was thought to be from diverticular disease.

In a recent report, Earley [*17*] said, "The mere presence of a neoplasm did not necessarily mean that it and not diverticular disease was the cause of hemorrhage."

Keith [*14*] states that he has been impressed with the frequency with which diverticula of the colon occurs in patients with severe melena and in whom no other etiological lesion could be demonstrated.

There are five main causes of colonic bleeding. They are diverticular disease, adenoma, hemangiomas, malignancy of the colon, and lipomas.

Polyps of the colon bleed severely at times especially if they have undergone malignant change and ulceration has occurred. Both polyps and diverticular disease may exist in the colon occasionally and it is difficult, if not almost impossible, to know the origin of the bleeding preoperatively. The bleeding point may be located at surgery, especially after the speciman is removed, but even this is difficult. Blood found in the lumen of the diverticulum is excellent evidence that it is the source of the bleeding. However, this evidence is not often found. Hemorrhage from diverticular disease may be extremely massive and the patient's blood count may fall extremely low.

Hemangiomas, especially small ones, must be looked for at surgery and ruled out. Even with the most careful search they may be overlooked, and they are sometimes found only at autopsy.

Carcinoma of the colon is usually discovered by sigmoidoscopy, palpation and roentgenographic studies. The bleeding is usually small in amount.

Among seven patients with lipomas of the distal colon seen at Emery University Hospital between 1949 and 1958, three patients had gastrointestinal bleeding.

Seabrook [*18*] also reported one case in which severe bleeding occurred from a diver-

ticulum of the cecum. This was proved by resection of the cecum and right colon.

TREATMENT

The treatment of bleeding from diverticular disease may be divided into two phases. First, conservative management and second surgical management. Forty-three patients were treated conservatively. Only twenty of the total of fifty patients required transfusions. The amount of blood used ranged from 500 cc. to 8,000 cc.

The amount of blood used was gauged by the severity of the bleeding as indicated by the hemoglobin value. Of the twenty patients, two had 5 gm. per cent, one had 6.8 gm. per cent, one had 8 gm. per cent, and five had 9 gm. per cent. Seven patients had 11 gm. per cent or less. The four remaining had more than 11 gm. per cent. There were six patients who needed only one transfusion of 500 cc., six received 1,000 cc., two were given 1,500 cc., one was given 2,000 cc. and two required 3,000 cc. Of the remaining three patients, one received 3,500 cc., one was given 5,000 cc. and one received 8,000 cc.

It seems logical to treat bleeding from diverticular disease of the colon conservatively when possible. It is almost impossible to pinpoint the bleeding lesion in the colon, at times, during surgical exploration. The diverticula may be scattered over the entire colon and it would be necessary to perform a complete colectomy as was performed by Waugh [19] and reported in a recent case report from the Mayo Clinic.

Therefore, the indications for conservative management in these cases can be based on the following facts (Table VI):

1. Turnbull [20] treated twelve patients conservatively. Keith and Rini [14] treated nineteen patients conservatively and fourteen of these required more than 1,000 cc. of blood.

TABLE VI
CONSERVATIVE TREATMENT

Series	Total No. of Patients	No. of Patients Transfused (1,000 cc. or more)
Good Samaritan Hospital	43	20
Turnbull	12	8
Keith and Rini	19	14
Stanton	14	9

Stanton [21] cared for fourteen patients and some required over 2,500 cc. of blood and were managed without surgery.

2. Forty-three of the fifty patients in our series were treated conservatively and they all survived.

3. Diverticular disease may extend over the entire colon and pinpointing the bleeding is most difficult, if not impossible.

4. Many of these patients are poor risks for surgery especially those in the older age group. Vascular problems and obesity are indications against operating.

5. In this series, operations were seldom performed upon the unprepared bowel.

6. The mortality rate may be as high as 40 to 50 per cent.

After the bleeding has stopped with conservative management roentgenograms of the colon should be taken. Sigmoidoscopy and air contrast studies should also be performed. The decision as to future treatment can then be made. If surgical treatment is indicated, the patient then can be carefully and most satisfactorily prepared for surgery. (Table VII.)

The second phase of treatment in bleeding

TABLE VII

Patient	No. of Transfusions Received	Erythrocyte Count (per cu. mm.)	Hemoglobin (gm. %)	Operation Performed
J. T.	6	1.5	5	
M. W.	2	3.06	5.6	
R. C.	16	2.21	6.8	Exploration and cecostomy
E. C.	4	3.10	8.0	
K. R.	3	3.60	9.0	
J. W.	2	3.42	9.5	
M. K.	7	3.76	9.7	Resection
H	1	3.47	9.8	
S	2	3.25	9.5	
P. K.	4	3.66	10.5	
K	2	3.9	11.4	
A. S.	3	3.81	11.7	
M. B.	1	4.44	12.7	
T. L.	2	4.4	12.1	
E. J.	1	4.34	12.5	
T. F.	10	2.3	6.0	Five operations
E. W.	1	4.48	12.2	
C. S.	1	4.71	14.0	

from diverticular disease is surgical management. In appreciable hemorrhage conservative treatment should be abandoned and laparotomy should be advised for patients who have massive colonic bleeding. Bargen [22] states, "I have seen several patients who have had massive intestinal bleeding and when surgery was performed, the only cause for the bleeding was a large open vessel in the diverticulum." The late Dr. Lahey [23] believed that when considerable hemorrhage occurred, conservative treatment should be abandoned and laparotomy advised. This form of treatment was given in seven of our twenty cases.

Of the seven patients who were operated upon in this series, one patient had a cecostomy, one a resection of the right colon, one a transverse colostomy, and four had a resection of the left colon with an end-to-end anastomosis.

CASE REPORTS

CASE I. R. C. is a man sixty-five years of age. Several years ago this patient had one episode of bleeding. He entered Good Samaritan Hospital at 4 A.M. He had bled for forty-eight hours and passed fresh blood. The bleeding was intermittent but at 12 A.M. on the third day the patient was bleeding so profusely that he was taken to surgery. The entire colon was filled with blood and it was impossible to determine the origin of the bleeding. A cecostomy was carried out and this was complicated by an obstruction in the small intestine and the patient died forty-eight hours later. Autopsy showed diverticular disease of the entire colon.

CASE II. M. K. was operated upon three days after admission to the hospital. The patient had gastrointestinal bleeding and presumedly diverticular disease in the cecal region. The operation was carried out three days later and a right colectomy and ileotransverseostomy was performed. The pathologic diagnosis was diverticulosis of the cecum and ascending colon. This patient's hemoglobin was 9 gm. per cent and the erythrocyte count was 3.7 million per cu. mm.

CASE III. T. F. entered the hospital because he passed bright red blood rectally. He became unconscious from loss of blood, the pulse was thready and he had considerable bloody diarrhea. The diagnosis was diverticulosis of the sigmoid and bleeding from the large intestine. The patient received ten transfusions, but the bleeding continued. A laparotomy was performed and endoscopy was used in three areas. The operating surgeon was unable to locate the bleeding vessel and a transverse colostomy was performed. The patient made an uneventful recovery with the exception of a wound dehiscence. Two months later the transverse colostomy was closed. No further surgery has been performed and the patient has remained in good health.

CASE IV. The patient entered the hospital complaining of colonic bleeding. The diagnosis was made of diverticulosis and diverticulitis of the left colon. Bleeding continued and the patient was operated upon. A segment of the left colon was removed. Pathological diagnosis was diverticulosis of the sigmoid and descending colon.

CASE V. A. J., a patient who was sixty-eight years of age, entered the hospital complaining of cramping pain and melena. The hemoglobin was 12 gm. per cent and the erythrocyte count was 3.8 per cu. mm. The initial diagnosis was diverticulitis of the sigmoid colon and a resection of the sigmoid area was performed. Pathologic diagnosis was diverticulosis of the descending colon. Three transfusions were given. This patient has remained well.

There were two other patients with bleeding from diverticular disease of the descending colon and the sigmoid area. The bleeding in these two cases was only moderately severe. However, resection was performed with uneventful recovery in both.

SUMMARY

I have presented our experience with fifty cases of bleeding due to diverticular disease out of a total of 250 patients treated during the ten-year period January 1948–January 1959.

In thirty of these patients there was a minimal amount of bleeding. Twenty of the fifty required at least one transfusion. This was 8 per cent of the total 250 cases.

Forty-three of these patients were treated conservatively and all made an uneventful recovery. There were seven patients who came to surgery, and one of these died. The remaining six patients are living and well today.

It seems that many writers believe that diverticulosis is the causative factor in bleeding as frequently or more frequently than diverticulitis. These two disease entities are commonly known and described by most authors as diverticular disease of the colon. Since diverticular disease of the colon is prone to cause bleeding, there is no doubt that elective resection should certainly be carefully considered at an opportune time in all cases.

In the older age group with diverticular disease of the colon the following facts seem reasonable:

1. Bleeding occurs in a percentage ranging from 4 to 47 per cent.

2. In the Good Samaritan Hospital series only twenty cases (8 per cent) required one or more transfusions.

3. Diagnosis should be made as accurately as possible.

4. Replacement of blood lost should be performed cautiously.

5. Conservative management is primarily indicated and is safer and satisfactory in most cases (forty-three of fifty) in the Good Samaritan Hospital series.

6. Mortality with immediate surgical exploration in the acutely bleeding patient may be as high as 40 to 50 per cent.

7. Treat each case as an individual problem.

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DISCUSSION

Henry N. Harkins (Seattle, Wash.): Dr. Howard has discussed a practical subject which is a fairly common entity, but in this particular instance it is a difficult problem to which I do not have any final answer.

We tabulated some statistics in our hospital with the assistance of Dr. John Stevenson. I will quote these because they essentially agree with Dr. Howard's figures. In each instance the incidence is a little higher.

For example, while the over-all incidence of bleeding in diverticular disease among Dr. Howard's patients was 20 per cent, ours (in a smaller series) was 25 per cent, but that is still in the range he mentioned (4 to 47 per cent).

Massive bleeding, which is subject to great variation according to who interprets the figures, occurred in 12 per cent of the patients in our series as opposed to 8 per cent in Dr. Howard's series. These figures are from the King County Hospital and therefore represent a slightly different type of patient.

Another interesting fact can be derived from our figures. If we divide the hemorrhages from the lower gastrointestinal tract into massive, moderate, and mild bleeding, we find that diverticular disease was definitely the most common cause of massive hemorrhage. In patients who had moderate bleeding it was close to the most common cause; whereas it definitely was not the most common cause of the milder hemorrhages.

I wish to comment on the difference in incidence and also in the nature of bleeding from diverticulosis and that from diverticulitis. Dr. Rives of Louisiana State University, has reported one of the largest series, and I have a very recent communication from him. He states, "In our series of 129 cases of massive melena, twenty-eight were attributed to diverticulosis, and (in our opinion) this was the second most frequent cause of massive melena without hematemesis."

Unlike our cases, his series of 129 cases of massive melena does include gastric causes. Duodenal ulcer is the most common, diverticulosis being the second most common, but if you consider the lower

intestinal causes alone, diverticulosis leads all the rest.

I also wish to comment on the difference in the incidence of bleeding from diverticulosis or from diverticulitis as to the extent of bleeding. In our opinion, massive hemorrhages are more common in diverticulosis, and possibly the milder hemorrhages are more common in diverticulitis. There is an analogy here to the cases of bleeding from the lower esophagus in peptic esophagitis. This is usually mild with persistent hemorrhage over a long period of time. Barrett's ulcer, a much rarer condition, causes bleeding more often. However, as Dr. DeVito of our department has reported, massive hemorrhage can occur even with peptic esophagitis.

Dr. Noer of Louisville has studied the vascular supply to the diverticula and showed that an unusual concentration of vessels (similar to a glomus) is present around the diverticula. These reports were made in a paper that he wrote some five years ago, and I think this explains anatomically what Dr. Howard was mentioning.*

Diagnosis and treatment are so intertwined that it is difficult to give an answer. It is certainly true that the bleeding from diverticulitis is an indication for operation on the affected part of the bowel, but the bleeding from diverticulosis is a much more difficult problem. As we all know, it is very difficult to localize bleeding from the colon even when it is caused by a polyp. In the case of diverticula, if they are multiple, it is even more difficult. While I would perform a blind partial gastrectomy on occasion, a blind colectomy should be approached with a little more circumspection, to say the least.

Another point is that the mere presence of bleeding does not differentiate between carcinoma and diverticulitis. Ponka, Brush and Fox* reported that the incidence of bleeding was 68 per cent in carcinoma and 25 per cent in diverticulitis. Goldman has just presented figures of 82 and 37 per cent in a series which is comparable to Ponka's. This substantiates the opinion that bleeding is not a criteria in differential diagnosis.

While we are now recognizing the importance of bleeding, we are in a position similar to that in the diagnosis of gastric ulcer and gastric carcinoma when there was a margin of diagnostic error. When the margin of error was 15 to 20 per cent, gastric resections were indicated more frequently. Similarly, in the present diagnostic controversy concerning diverticulitis and carcinoma of the colon, a more aggressive attack may be indicated from this standpoint alone.

MARTIN A. HOWARD (closing): I wish to thank Dr. Harkins for his very able discussion of my paper. It is interesting to note that the incidence of bleeding from various series has been as great or greater than those at Good Samaritan Hospital.

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Chorionepithelioma of the Stomach

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CHORIONEPITHELIOMA is a relatively uncommon malignant tumor of the trophoblast, most frequently originating within the uterus, less frequently encountered in the oviducts, ovaries and male gonads, and seldom in extragenital sites such as the mediastinum, retroperitoneum and pineal gland [*12*]. We treated a patient with extragenital chorionepithelioma of the stomach. Sixteen similar cases were found in the literature. The description of our case and a review of the pertinent literature form the basis of this report.

CASE REPORT

A seventy-seven year old white man visited our office on February 18, 1956, complaining of indigestion after eating, which he had experienced for a period of three days. When seen on March 22, 1956, he had lost 6½ pounds and complained of nausea and weakness which occurred in the morning. He stated that he had been taking "a half ounce" of milk of magnesia or Amphojel® every two hours for the "hurting in his stomach." On July 23, 1956, he stated that his left nipple had been painful upon contact with hand or towel for three years. It was slightly fuller than the right. He was given 100 mg. of testosterone intramuscularly for this symptom. A few days later both breasts became sore and remained sore. On December 14, 1956, he stated that he had had nausea associated with loose stools for three to four days.

Inquiry into his past history revealed that he had experienced burning at the level of the navel fifteen to forty-five minutes after eating during October 1953. It had been relieved by water and food. He had had similar episodes intermittently since the age of twenty-one years. Roentgenograms of the stomach taken in 1945, and again in 1947, had been reported as negative.

Roentgenograms taken on October 16, 1953, had revealed an indefinite ulcer crater at the junction of the fundus with the body along the lesser curvature of the stomach. Another roentgenographic study on December 4, 1953, had been reported as negative. He had been treated for hypertensive cardiovascular disease (blood pressure of 200/100 mm. Hg) and had been comfortable until October 1954, when he had been referred to a urologist because of progressive difficulty in urination associated with chills, fever and pyuria. A diagnosis of benign prostatic hypertrophy, azotemia, and bilateral hydronephrosis and hydroureter, secondary to obstruction at the neck of the bladder was made. On December 13, 1954, a retropubic prostatectomy and bilateral partial vasectomy had been performed. The pathologist reported a benign hypertrophy of the prostate gland.

Shortly after the onset of symptoms the patient was advised to have an examination of the gastrointestinal tract but refused for financial reasons. On January 2, 1957, he was admitted to the hospital for study. Physical examination revealed an emaciated white man weighing 127 pounds and measuring 69 inches in height. His skin was dry and warm, and he was edentulous. The chest was clear to percussion and auscultation. The right breast was enlarged and tender. There was no enlargement of the heart. The heart rate was 72 with frequent extrasystoles. There were no murmurs. Blood pressure was 140/60 mm. Hg. The liver was palpable 3 to 4 fingerbreadths below the costal margin. A left inguinal hernia was present. Rectal examination revealed a small prostate gland. The genitalia were normal.

Roentgenograms of the stomach were reported as showing a filling defect in the cardiac portion of the stomach, which involved the greater curvature and probably the lesser curvature.

Laboratory studies revealed 12.3 gm. per cent of hemoglobin; 11,000 leukocytes per cu. mm.; the differential count revealed 83 per cent neutrophils, of which 80 per cent were segmented and 3 were band cells; there were 5 lymphocytes and 12 monocytes. The red blood cells appeared normal. The blood platelets appeared normal in number. Urinalysis revealed a specific gravity of 1.016, an acid reaction, no sugar, and a trace of albumin. Microscopic examination of the urine revealed 50 to

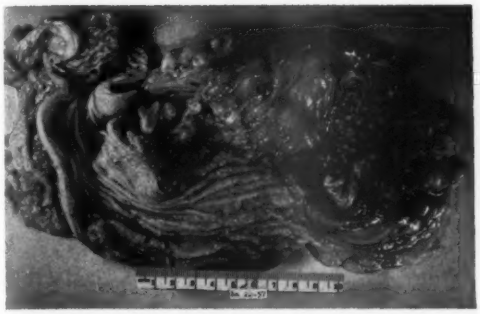

FIG. 1. Stomach opened longitudinally showing fungating tumor near cardia on right.

FIG. 2. Cut surface of right lobe of liver revealing two types of metastases—the solid white (carcinoma) and the spongy hemorrhagic red (hemorrhagic chorionepithelioma).

70 white blood cells per high power field and 10 to 20 red blood cells per high power field.

A laparotomy was performed on January 8, 1957, at which time a large mass was found involving the greater curvature of the fundus of the stomach. The mass was approximately 8 cm. in maximum diameter. The liver contained large nodules, within which there appeared to be dilated vessels filled with old blood clots. A biopsy of one of these nodules was taken for microscopic examination. The pathologist reported the lesion as metastatic choriocarcinoma of the liver. Following this, a Friedman test was performed and was reported as strongly positive for chorionic gonadotropin.

The patient's course was rapidly downhill. Bronchopneumonia developed, and he died on February 2, 1957.

Postmorten Examination. On external examination there was evidence of emaciation, enlargement of the right breast, a recent abdominal incision and an old prostatectomy scar. On primary incision, there were peritoneal and pleural fibrous adhesions. There was a small bilateral pleural effusion. A firm nodule measuring 2.5 cm. in diameter, in contiguity with the aorta just below the arch proved to be hyperplastic lymphatic tissue on histologic examination.

At the level of the cardia of the stomach on the greater curvature there was a grayish-red, fungating and ulcerated tumor. It measured 7 cm. in diameter and was centrally depressed and peripherally firm and raised. (Fig. 1.) The tumor extended through all layers of the wall of the stomach and produced a subserous bulge of 3 cm. Its cut surfaces were deep red to blue and moist.

The liver was enlarged and weighed 3,030 gm. The external and cut surfaces revealed numerous tumors of various sizes, the largest being 7 cm. in the greatest dimension. (Fig. 2.) Principally, these were deep red and soft, and some had necrotic centers. Also, there were two relatively small, grayish-tan nodules, the larger being 2.5 cm. in the greatest dimension. Otherwise, the liver had a pale brown color with a normal lobular architecture.

The lungs were edematous and the seat of bronchopneumonia. In addition to a 1 cm. calcific nodule, there was a 1.5 cm., reddish-tan, soft subpleural nodule in the right lower lobe.

The testes were grossly normal. In the right epididymis there was a 5 mm. cyst having the characteristic structure of a spermatocele.

There were findings of atherosclerosis involving the coronary arteries and aorta, hyalocapsulitis of

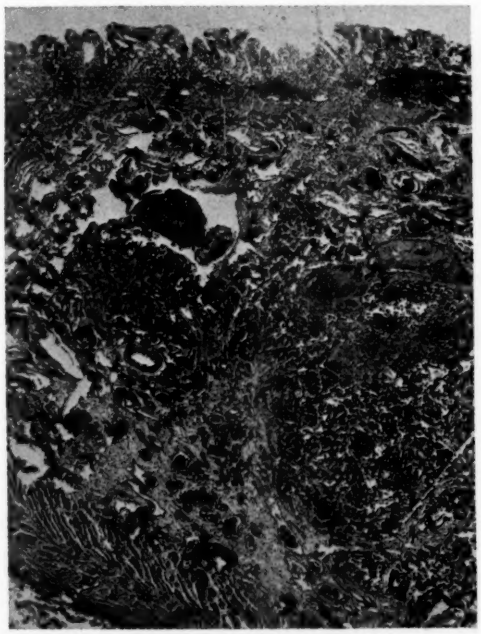

FIG. 3. Wall of stomach invaded by tumor. Original magnification × 20.

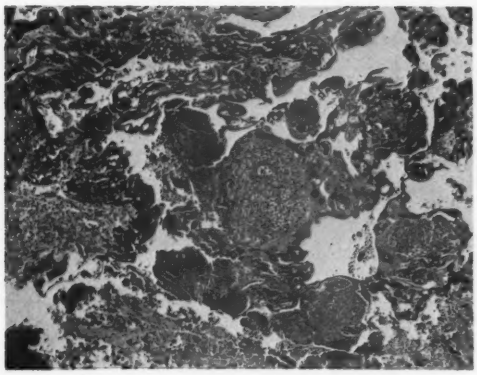

FIG. 4. Photomicrograph showing chorionepithelioma pattern in stomach tissue. Original magnification × 55.

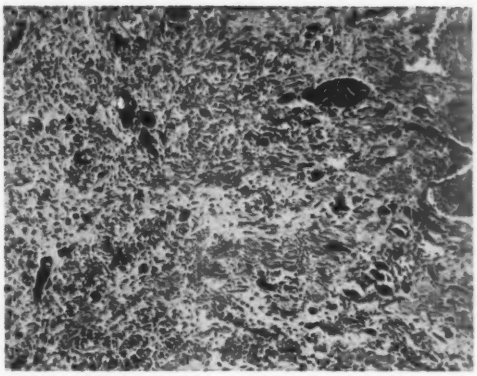

FIG. 5. Photomicrograph showing invasive carcinoma pattern in stomach tissue. Original magnification × 85.

the spleen, nephrolithiasis on the right side, bilateral hydronephrosis and nephrosclerosis and a non-neoplastic residuum of the prostate. No gross abnormalities were found in the brain, pineal gland or pituitary.

Microscopic Examination. Routine hematoxylin and eosin and, selectively, periodic-acid Schiff and mucicarmine stains were employed on material fixed in formalin and embedded in paraffin.

The gastric tumor was entirely embedded, with some of the paraffin blocks being examined at step levels. The tumor invaded all layers of the wall of the stomach and occupied the lumina of some thin-walled, frequently dilated blood vessels and lymphatics. (Fig. 3.) There was broad ulceration of the mucosa, with the mucosa at the margin of the tumor being undermined and elevated. There was extensive necrosis of the tumor with cyst-like spaces which were filled with necrotic debris. The stroma was scanty. At the periphery, epithelial tumor cells infiltrated the tunica muscularis propria without tumor stroma. The tumor had two major histologic patterns. The dominant pattern was characterized by cells which were chiefly large and irregular in shape with poorly defined boundaries and varying quantities of amphophilic to faintly eosinophilic cytoplasm. The nuclei varied in size with some gigantic and very bizarre forms. Hyperchromatism and pleomorphism and frequent large nucleoli were conspicuous. There were relatively rare mitotic figures. Some of the tumor cells were multinucleated with as many as a score of nuclei in one cell. Mitotic activity was not observed in these cells. Some of this neoplastic epithelium was vacuolated or "honeycombed" in appearance. These tumor cells tended to line variously sized spaces and blood vessels. The large spaces contained necrotic debris. Some small spaces contained blood cells. This picture duplicates that of chorionepithelioma exactly. (Fig. 4.) The minor pattern was characterized by small sheets of somewhat smaller cells with distinct boundaries and pale, frequently vacuolated cytoplasm. The nuclei here were hyperchromatic with distinct nucleoli but less large and bizarre than those previously described. Mitoses were more numerous here. Multinucleated giant cells were not present in these areas. This

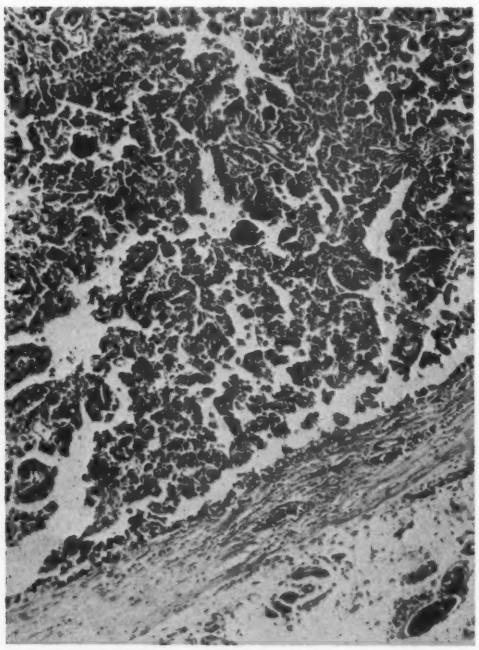

FIG. 6. Perigastric lymph node showing metastatic carcinoma with psammoma bodies. Original magnification × 75.

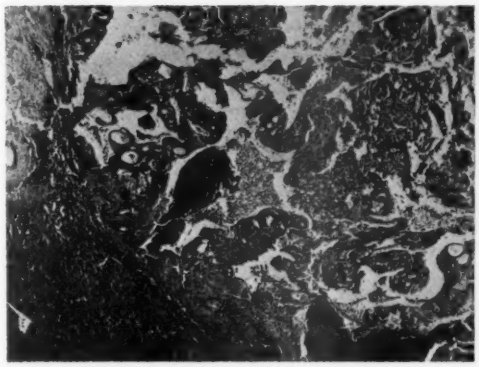

FIG. 7. Photomicrograph showing metastatic chorionepithelioma pattern in liver tissue. Original magnification × 35.

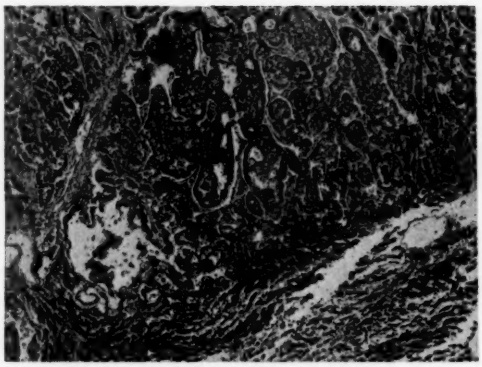

FIG. 8. Photomicrograph showing carcinoma pattern in liver tissue. Original magnification × 35.

picture is that of anaplastic carcinoma and indistinguishable from other undifferentiated gastric carcinomas. (Fig. 5.) No Schiff-positive or mucicarminophilic material was demonstrated in these cells. No structures identifiable as glands were formed by the tumor cells.

Twenty demonstrable perigastric lymph nodes, both from the lesser and greater curvatures, were embedded for histologic study. Three of these nodes showed almost complete replacement by tumor. In two of the nodes there was a solid to papillary, nonnecrotic growth comprising tumor cells similar to those of the pattern of undifferentiated carcinoma in the gastric tumor. In one of these nodes psammoma bodies were present in small number. (Fig. 6.) In the third node involved by tumor, there was fairly extensive necrosis and here the pattern simulated the chorionepithelioma pattern of the gastric neoplasm.

The metastases in the liver also presented two patterns. Corresponding to the predominant deep red, soft tumors were necrotic growths with a pattern of chorionepithelioma. (Fig. 7.) Corresponding to the grayish tan nodules were growths having the carcinoma pattern. (Fig. 8.) Here, however, a few crude glandular structures were present, stroma was more abundant and a few psammoma bodies were seen.

The reddish tan pulmonary nodule of the lower lobe of the right lung had a somewhat mixed pattern with features of chorionepithelioma and undifferentiated carcinoma.

Each testis was totally embedded and the sections failed to show evidence of tumor. There was some slight focal atrophy with fibrosis compatible with the patient's age, but no remarkable scar was encountered. The Leydig cells were normal in number and distribution.

The right breast had the histologic structure of gynecomastia. (Fig. 9.)

The pituitary revealed an increased number

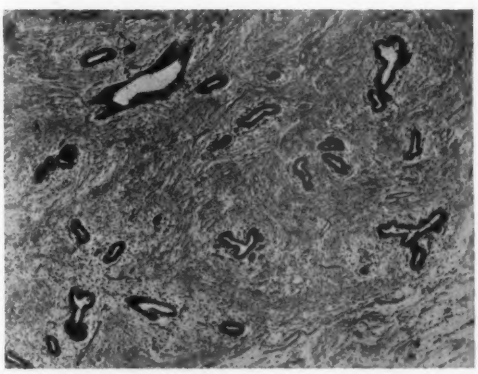

FIG. 9. Photomicrograph showing gynecomastia in tissue from the right breast. Original magnification × 35.

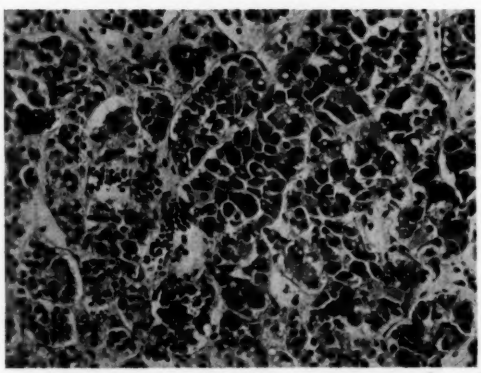

FIG. 10. Pregnancy cells in the anterior pituitary. Original magnification × 250.

TABLE I

SUMMARY OF CASE REPORTED AND THOSE FROM LITERATURE

Case No.	Author	Year Published	Sex, Age	Location of Primary Tumor	Location of Metastases	Type of Metastases	Condition of Genitalia	Hormonal Test
1	Davidsohn [3]	1905	M, 44	"Sac" the size of a child's head between stomach and liver; adenocarcinoma of pylorus of stomach, measuring 7 cm.	Liver, lymph nodes	Red and white in liver; histologically carcinoma and chorionepithelioma	Normal testes	None
2	Bonney [20] (cited by Symeonidis)	1906	M, 69	Deep red tumor with the appearance of chorionepithelioma on greater omentum connected with stomach on greater curvature; no actual gastric tumor described	Liver	Dark red chorionepithelioma in liver	Normal testes	None
3	Helmholz [5]	1907	M, 40	Histologic chorionepithelioma measuring 10 cm. on antrum of stomach	Lymph nodes, liver, spleen, lung; extension to pancreas and colon	Yellowish and reddish-yellow in lymph nodes; yellow and reddish-brown in liver; histologically chorionepithelioma	Normal testes	None
4	Risel [16]	1907	F, 40	Ulcerated mucinous carcinoma of pylorus of stomach	Liver; extension to colon	Red and yellow-white or yellow-green nodules in the liver; red metastases resembled chorionepithelioma histologically	Small uterus with small subserous myoma; ovaries small and hard	None
5	Risel [16]	1907	F, 52	Stenosing adenocarcinoma of pylorus of stomach measuring 3 by 3.5 cm.	Lymph nodes, liver, retroperitoneum, lung; extension to duodenum	Yellow-white and red in lymph nodes; white-yellow and red in liver; hemorrhagic metastases in lung showed hemorrhagic red tumor histologically resembling chorionepithelioma; white tumor of liver was carcinoma	Normal uterus and ovaries; Para IX with last delivery sixteen years prior to admission	None
6	Stewart [17]	1913	F, 49	Perforated, ulcerated adenocarcinoma of stomach	Lymph nodes, lung, liver and peritoneum	White in lymph nodes; resembled placenta in omentum; white histologically resembling carcinoma; and red histologically resembling chorionepithelioma in liver	Normal uterus and ovaries	None
7	Koritschoner [10]	1920	F, 61	A 3.5 by 4.5 by 2.5 cm. tumor of antrum of stomach; histologically chorionepithelioma and questionable carcinoma	Lung, liver, spleen	Yellow to hemorrhagic nodules in liver; white hemorrhagic node in lung; dark colored and seed-like histologically resembling chorionepithelioma in spleen	Bloody nodules in uterus but no evidence of choriocarcinoma; normal delivery twenty-two years prior to admission	Positive Abderhalden (proteinase reaction)
8	Stoy [20] (cited by Symeonidis)	1921	M, 40	Questionable primary tumor of liver with questionable metastasis to stomach; gastric tumor, histologically chorionepithelioma	Questionable primary tumor, liver; questionable metastases to stomach, spleen, intestine, portal nodes	Chorionepithelioma	Normal testes	None
9	Herms [20] (citing Ceelen) (cited by Symeonidis)	1924	M, 60	Gastric carcinoma	Widespread	"Chorionepithelioma-like"	Normal testes	None

TABLE 1 (*Continued*)

SUMMARY OF CASE REPORTED AND THOSE FROM LITERATURE

Case No.	Author	Year Published	Sex, Age	Location of Primary Tumor	Location of Metastases	Type of Metastases	Condition of Genitalia	Hormonal Test
10	Pick [13]	1926	M, 52	Ulcerating carcinoma with chorionepithelioma of the pylorus of stomach	Liver, lymph nodes, lung	Identical with chorionepithelioma in liver	Normal testes	None
11	Gaertner [5]	1938	M, 54	Pyloric adenocarcinoma	Liver, pancreas, lungs	White and red tumors in liver; yellow, red and gray in pancreas; red in lungs; white (carcinoma), red (chorionepithelioma) in liver; chorionepithelioma in lung; mixed chorionepithelioma and carcinoma in pancreas	Normal testes	None
12	Gaertner [5]	1938	M, 37	Carcinoma on greater curvature of stomach with structure histologically resembling choriocarcinoma	Liver	Red to yellow in liver; histologically chorionepithelioma	Normal testes	None
13	Hartz and Ramirez [7]	1953	M, 28	Ulcerating tumor of antrum of stomach with histologic structure of mucinous carcinoma, chorionepithelioma and teratoid tumor	Nodes, liver, lung	Hemorrhagic tumor in lymph nodes, liver and lung; histologically chorionepithelioma in liver and lung	Testes not examined	None
14	Stout [19]	1953	F, 76	Fungating carcinoma of stomach, including mucosa and submucosa; outer stomach with musculature between, chorionepithelioma	Questionable pulmonary metastasis (patient died; no autopsy)		Hysterectomy for adenomyosis thirteen years previous to admission	Positive Friedman after gastrectomy
15	Voss [21]	1954	M, 70	"Palm-sized," white to hemorrhagic tumor on lesser curvature of stomach below cardia histologically including polyp, carcinoma and chorionepithelioma	Liver, blood vessels, lungs, lymph nodes	Hemorrhagic to white in liver; hemorrhagic nodes in lung; histologic carcinoma in gastric nodes, lymphatics and liver; chorionepithelioma in liver and lung	Normal testes	Positive Aschheim-Zondek following exploratory laparotomy with biopsy of liver; positive turtle test for chorionic gonadotropin with serum obtained at autopsy
16	Goder [6]	1958	F, 72	Tumor of cardia of stomach measuring 6 by 5 cm. with hemorrhagic nodules, histologically carcinoma and chorionepithelioma	Lymph nodes, liver, omentum	Histologically chorionepithelioma	Normal uterus and ovaries; no history of abortions or deliveries	Positive male frog test with liver tissue; inconclusive Aschheim-Zondek test on tissue from autopsy
17	Authors' case	1960	M, 77	A fungating, ulcerated tumor (7 cm.) of greater curvature of stomach with carcinoma plus chorionepithelioma	Lung, liver, lymph nodes	Red and white nodules in liver; red nodule in lung; carcinoma in lymph nodes and liver; chorionepithelioma in lymph nodes, liver and lung	Normal testes	Positive Friedman

(over 60 per cent) of acidophilic cells with abundant coarse, granularity of the cystoplasm and elongation of some of the cells similar in appearance to pregnancy cells. (Fig. 10.)

The thyroid showed no distinctive histologic change.

Review of the slides of the tissue removed at the time of prostatic resection revealed nodular hyperplasia but no neoplasia.

COMMENTS

It was evident that we were dealing with a carcinoma and a tumor grossly and histologically compatible with chorionepithelioma involving the stomach, with obviously metastatic foci of carcinoma and chorionepithelioma in lymph nodes, liver and lung. In support of the actual chorionepitheliomatous nature of the tumor were a strongly positive test for chorionic gonadotropin, gynecomastia, and pituitary change such as may be seen in chorionepithelioma. Complete autopsy excluded a genital or extragenital site other than the stomach as the primary tumor. No significant testicular cicatrix which, according to Prym [*14*], may mark the site of healed or regressed chorionepithelioma, was demonstrated.

Sixteen remarkably similar cases were found in the literature and are summarized in Table 1. With the exception of Case 2, each had either a carcinoma, a chorionepithelioma or a mixture of chorionepithelioma and carcinoma of the stomach. In Case 2, there was a chorionepithelioma of the greater omentum attached to the stomach but no actual gastric neoplasm was described. In three of the cases (3, 8 and 12), the gastric tumor was described as chorionepithelioma. In six cases (1, 4, 5, 6, 9, 11), the gastric tumor presented the features of carcinoma and chorionepithelioma. In two cases (1 and 14), there was a carcinoma of the stomach with a nearby but probably related chorionepithelioma. In Case 1 there was an omental sac of chorionepithelioma attached to a carcinoma of the pylorus. In Case 14 there was a chorionepithelioma on the outer surface of the stomach separated from a gastric carcinoma by musculature.

A primary genital or gonadal neoplasm was excluded in all but two cases: in Case 13 the testes were not examined; in Case 14 a hysterectomy had been performed for adenomyosis thirteen years before and no autopsy was performed, leaving the gonadal status in question. In Case 15 microscopic sections from the entire testes excluded any scar, such as may be the stigma of a healed or regressed chorionepithelioma, as described by Prym [*14*].

In all cases there was metastatic tumor. In Case 14 pulmonary metastases were diagnosed clinically, but no autopsy was performed. In five cases (1, 5, 6, 11, 15), there were metastatic foci of both carcinoma and chorionepithelioma which, characteristically, were described as "white" and "red" (hemorrhagic) respectively. In eight cases (2, 3, 7, 8, 9, 12, 13, 16), only chorionepithelioma metastases were present. In Cases 4 and 10, only chorionepithelioma metastases were noted, but the description is incomplete and carcinomatous metastases may have been present.

In the only three cases (14, 15, 16) in which the biologic test for chorionic gonadotropin was performed, it was positive. In one case (15) gynecomastia was described.

It is apparent that there are a significant number of cases similar to ours. The common denominator is extragenital chorionepithelioma or, for the sake of argument, pseudochorionepithelioma, in gastric or perigastric neoplasms and/or their metastatic sites. In Case 1 the perigastric chorionepithelioma was probably related to the intragastric neoplasm, while in Case 2 this relationship was only a possibility.

Is this tumor an actual chorionepithelioma or pseudochorionepithelioma? This is a problem which has plagued other observers of this tumor and does not have a simple solution. Risel [*16*], Stewart [*17*], Borrmann [*2*] and Symeonidis [*20*] believed that the tumor only resembled a chorionepithelioma and that it represented a peculiar degenerative change of a gastric carcinoma. Others, including Davidsohn [*3*], Rich [*15*] (after reviewing the slides from Case 3), Koritschoner [*10*], Pick [*13*], Hartz and Ramirez [*7*], Stout [*19*], Voss [*21*], and Goder [*6*], regarded the tumor as a bona fide chorionepithelioma.

The chorionepithelioma in our patient appears morphologically genuine since all of the other pathologists, including Arthur Purdy Stout [*18*], to whom the slides have been shown, regard it as morphologically consistent with chorionepithelioma. Further support for the diagnosis of chorionepithelioma is afforded by the strongly positive Friedman reaction, gynecomastia and pituitary change [*9*].

Since we are restricted by the morphologic and functional characteristics of a tumor for the

purposes of classification, and since this tumor is morphologically and functionally compatible with chorionepithelioma, we are left with no alternative but to classify it as such. To classify these cases as pseudochorionepithelioma would leave us in the untenable position of ignoring morphology and function in classifying any tumor.

What is the genesis of this neoplasm? Davidsohn [3] suggested that its origin was in abdominally displaced gonadal anlage. Koritschoner [10] postulated a long-delayed (twenty-two years) metastasis of a hypothetical intrauterine chorionepithelioma. Stout [19] assumed that it was a metastasis. Hartz and Ramirez [7] and Goder [6] postulated that the origin was in gastric teratoma. Voss [21] believed that the origin was in a totipotential cell of primary gonadal type occurring in a gastric polyp. Pick [13] hypothesized a retrodifferentiation or opisthoplasia of carcinoma cells with retrograde increase of embryonal potentialities to the level of embryonal ectoderm with the ability to form trophoblast. He made an analogy to the metaplasia of entodermal epithelial structures, such as the mucosa of the gallbladder and bronchi, to form epidermoid ectodermal tumors.

Extragenital or nongestational chorionepithelioma is usually thought to arise in a teratoma or displaced germinal epithelium [4]. Save that *a priori* chorionepithelioma may be regarded as evidence of teratoma, no evidence of teratoma of the stomach existed in any of the cases. In the case of Hartz and Ramirez [7], the "teratoid tumor" component may be readily interpreted as poorly differentiated carcinoma. No evidence for teratoma, which was postulated, exists in the case of Goder [6]. Germinal epithelium from the embryonic urogenital ridge may explain certain retroperitoneal and mediastinal chorionepitheliomas. However, germinal epithelium as a component of a gastric neoplasm is too farfetched an explanation for serious consideration.

Gastric carcinoma was present in thirteen of the cases, including ours. It is extremely unlikely that gastric carcinoma in three-fourths of these cases was merely coincidental and that chorionepithelioma was a metastasis, with the primary tumor postulated as an unproved and generally well excluded chorionepithelioma arising at other sites. It is more logical to explain the chorionepithelioma in terms of the condition with which it is most frequently associated in these cases, that is, gastric carcinoma. The tumor was located in the antrum or pylorus in ten of the seventeen cases. The sex distribution is eleven men to six women. The ages ranged from 28 to 77 years, and the average age was 54 years. Although the number of cases may not be significant statistically, these figures and the location coincide with those given for gastric carcinoma [1,19].

We are forced to conclude, in agreement with Pick [13], that gastric carcinoma cells may retrodifferentiate to form tumor morphologically and otherwise indistinguishable from chorionepithelioma. The chorionepithelioma pattern of this gastric neoplasm may completely dominate the picture so that the carcinomatous basis or origin of the tumor may be hidden or completely lost. The event is analogous to the development of adenocanthoma or squamous cell carcinoma of the stomach [11]. Ten of the seventeen patients had metastases to the liver of the white (carcinoma) and red (chorionepithelioma) types. From a purely practical standpoint, the finding of a gastric neoplasm of unusual hemorrhagic appearance with white and red metastases in the liver should alert the surgeon and the pathologist to the probable presence of this tumor.

SUMMARY AND CONCLUSIONS

On the basis of the study of our case and others cited from the literature, it is clear that there is a rare gastric neoplasm which has features which are indistinguishable, morphologically and functionally, from chorionepithelioma which arises in genital or accepted extragenital sites.

Acknowledgment: We wish to express our appreciation to William H. Kern, M.D. for his aid in translating original articles and to Mr. Lloyd Matlovsky for the preparation of the microphotographs.

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DISCUSSION

Henry N. Harkins (Seattle, Wash.): This is an unusual syndrome, not a mere anatomic variety of a rare tumor. Furthermore, the fact that Dr. Regan's case is only the third to be reported with determination of chorionic gonadotropin demonstrates the unexplored endocrine and physiologic indications of this syndrome.

Also, the dual nature of the tumor, with its white and red metastases, provides additional interest.

At first glance, the easiest solution seems to be to explain these as pseudochorionepitheliomas. However, on reading Dr. Regan's manuscript and on surveying the list of pathologists who have definitively stated that these are true chorionepitheliomas, it seems idle for me to dispute this viewpoint.

I was tempted to theorize regarding the origin of these tumors, and believed that they might represent a type of target organ failure. For example, the testicular atrophy or in some other cases the lifeless scars in the testes (described by Prym), with corresponding compensatory pituitary stimulation of another target organ, seemed logical. However, some of the selected cases were in young individuals with testes which proved to be normal, and there is no evidence that in this particular respect the stomach is a target organ. Thus I am forced to agree with Dr. Regan that the Pick theory of retrodifferentiation of gastric carcinoma cells is the most likely explanation of the origin of these tumors.

Dr. Regan has presented a syndrome with interesting anatomic, pathologic and physiologic implications. He diagnosed a case in a former patient of Dr. Helmholtz' at Johns Hopkins fifty-one years later by merely looking at the illustrations.

Frederic P. Shidler (Menlo Park, Calif.): In surgical pathology we have emphasized to students that the appearance of gynecomastia in the adult male, whether it be unilateral or bilateral, should be followed by thoughtful investigation such as palpation of the testes or a careful scrutiny of his liver, since this event represents an unusual reaction of the breast to estrogen or gonadotropic hormone of some kind.

A third source for gynecomastia is the concealed, obscure, chorionepithelioma of the stomach. I do not think that this patient was curable. Perhaps the diagnosis could have been established sooner if urinary hormonal assays had been performed.

I wish to ask Dr. Regan whether or not the left breast showed changes of gynecomastia similar to those in the right breast? If only one responded to the hormone, it becomes even a more exotic tumor than you have described.

James F. Regan (closing): In answer to Dr. Shidler's question regarding gynecomastia, I purposely mentioned in the history that the left breast had bothered the patient about seven months before he died, and that he said the breast had been tender for about three years.

It seemed a very peculiar coincidence that both breasts became sore after he was given testosterone by his internist, and that the right breast became more tender than the left and was larger at the time of his death. Microscopic examination of the tissue presented the appearance of gynecomastia. We wondered if a mistake had been made in the recording of the original history and physical findings.

On December 1, 1958, we received the following letter from Arnold R. Rich, M.D., of the Department of Pathology, The Johns Hopkins University: "I have examined the sections of tumor reported by Dr. Helmholtz in 1906, about which you inquire. It is indeed clearly a chorionepithelioma as you recognized from the illustration in Helmholtz' paper. It is highly interesting you have a similar case." The diagnosis was made over fifty-one years after the death of the patient.

Clinical Experiences with Preperitoneal Hernial Repair for All Types of Hernia of the Groin

WITH PARTICULAR REFERENCE TO THE IMPORTANCE OF TRANSVERSALIS FASCIA ANALOGUES

LLOYD M. NYHUS, M.D., ROBERT E. CONDON, M.D. AND HENRY N. HARKINS, M.D.,
Seattle, Washington

ANY operative approach in inguinofemoral hernias, to be practical, must intrinsically allow for immediate repair of indirect, direct, or femoral hernias. The concept of approaching hernias of the groin from inside the inguinal abdominal floor preperitoneally rather than from outside is generally attributed to Cheatle [2,3] and Henry [6]. Through succeeding decades this concept has failed to gain widespread acceptance, except possibly in the treatment of femoral hernia. Applicability of this approach to the repair of inguinal hernia, direct and indirect, has been lacking. Thus, the preperitoneal approach (from within) appears not to fulfill the criteria of a practical approach, and thus seems doomed to minimal use in our operative armamentarium. This assumption is, however, not correct. It has not been recognized that tissues are available in the preperitoneal approach for simple, yet apparently adequate, repair of each type of hernia of the groin. This apparently radical statement is made subsequent to our experience in the anatomic laboratory and the utilization of this approach for the repair of 213 separate inguinal and femoral hernias in 150 patients. The key to success of this preperitoneal technic is a thorough understanding of the transversalis fascia and its analogues, combined with proper utilization of this anatomic structure for repair, whether the hernia be femoral, direct, or indirect inguinal.

SURGICAL ANATOMY OF THE TRANSVERSALIS FASCIA AND ITS ANALOGUES

The abdominal wall is a multilaminar structure consisting of musculoaponeurotic layers ensheathed in their fascias, together with an internal (preperitoneal) and an external (subcutaneous) layer of fat, and limited by an internal endothelium (peritoneum) and an external epithelium (skin). Disruption, relaxation, or stretching of one or more of these abdominal laminas gives rise to a hernia. The essential object of a hernial repair is a restoration of the anatomy to its normal laminar situation. This anatomically and surgically rational conception of herniorrhaphy has been widely accepted and practiced, particularly with regard to hernias of the groin.

In the groin, inguinofemoral hernias all have in common the fact that each consists of a breakdown in that lamina of the abdominal wall known as the transversalis fascia. Direct inguinal hernia is a breakdown of transversalis fascia where it forms the posterior wall of the inguinal canal; indirect inguinal hernia is a disruption of the transversalis fascia lamina at the margins of the internal (abdominal) inguinal ring; it may progress to also involve the posterior wall of the inguinal canal. A femoral hernia is a disruption of structures of the transversalis fascia lamina at the orifice of the femoral canal. In every case of inguinofemoral hernia, then, the basis of repair must be an anatomic restoration of the continuity and integrity of the disrupted transversalis fascia lamina in the groin.

Our adherence to the principle of restoration of anatomic integrity of transversalis fascia in groin herniorrhaphy does not imply an unalterable commitment to utilize only transversalis fascia in the repair. However, it does imply that the structures utilized for repair lie

within the transversalis lamina of the inguinal region.

It is sometimes found that the true transversalis fascia is a veil, a thin filmy layer totally incapable of retaining a suture. Nonetheless, this layer still forms the essential guide to repair since it defines the lamina within which repair should be carried out. The structures which are often actually utilized in the herniorrhaphy are a group of ligamentous and aponeurotic structures closely associated with the transversalis fascia; we have termed these structures transversalis fascia analogues. They are capable of retaining heavy sutures and provide the strength required for the reapproximation and restoration of continuity of the transversalis fascia. This distinction between transversalis fascia, proper, and transversalis fascia analogues has been made in order to help clarify our conception of the principle of laminar repair.

As a part of this study, the normal anatomy of the inguinal region was reviewed in the autopsy room. Dissections were carried out in twenty male bodies, all without hernia (forty inguinal areas). The fresh material we utilized has considerable advantage to the dissector over the fixed cadaver material available in the anatomic laboratory. Tissue densities approximate those of the living patient; areolar tissue dissects as areolar tissue and is easily distinguished from tougher, stronger fascial and ligamentous structures. Artifacts induced by the fixing process are eliminated.

Transversalis Fascia

Transversalis fascia is the investing fascia on the inner surface of the transversus abdominis muscle; it covers that muscle and its aponeurotic tendon of insertion, forming the internal leaf of the muscle sheath.

It is important to remember that transversalis fascia is not an isolated structure, but part of the continuous sheet of fascia which encircles the entire peritoneal cavity, the endoabdominal fascia. Therefore, transversalis fascia, which covers transversus abdominis muscle, is continuous with iliacus and psoas fascia laterally and quadratus fascia posteriorly. Medially, it is continuous with the posterior investing fascia of the rectus abdominis muscle. Inferiorly, it inserts into Cooper's ligament; this relationship is particularly obvious medially and along the pecten of the pubis. However, transversalis fascia does not simply end there; it proceeds inferiorly to become continuous with the levator and obturator fascias.

The relationships of transversalis fascia at the internal (abdominal) inguinal ring is an unsettled matter. Anson et al. [*1*], McVay et al. [*11,12*], Donald [*4*] and Griffith [*5*] demonstrate a tubular projection of transversalis fascia covering the spermatic cord distal to the internal ring, which is designated internal spermatic fascia. Tobin and associates [*17*], on the other hand, conclude from their careful dissections that transversalis fascia closes over the internal inguinal ring, but does not extend over the cord as a definite layer of fascia. These previous studies by dissection have been carried out using fixed cadavers and there are valid objections to such minute dissections involving embalmed connective tissue because of the changes which occur in their texture. We are currently reinvestigating this matter utilizing fresh cadaver material. Suffice it to say, for the present, that in the presence of a long-standing indirect inguinal hernia there is a definite layer of condensed connective tissue located internal to the cremasteric muscle-fascia layer and external to the vas and vessels, which traditionally has been designated as internal spermatic fascia.

Transversalis Fascia Analogues

Perhaps, fortuitously, or because of the stresses to which it is subjected, the transversalis fascia in the hernial regions of the groin undergoes modifications leading to the formation of fascial condensations within it. These ligamentous thickenings together with contributions from adjacent muscular aponeuroses, form a group of structures we have termed transversalis fascia analogues. (Fig. 1.) They have, in common with all structures in the groin, a great deal of variability in regard to form and strength. Some are more constant and more important in hernial repair than others.

1. Transversalis Fascia as an Analogue. As noted previously, transversalis fascia in the normal person is usually not the strong resilient tissue of which a solid hernial repair is constructed; indeed, it may be nothing more than a film of connective tissue bridging the space between the ligaments which form its analogues. In the presence of a hernia, however, and particularly with a direct hernia which has been present for more than a few months, the transversalis fascia becomes markedly thick-

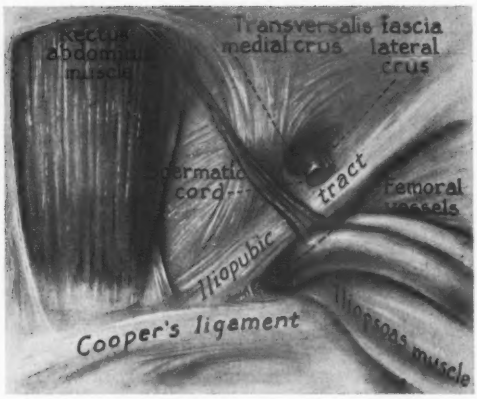


FIG. 1. Anatomy of transversalis fascia as seen from preperitoneal approach.

ened at the internal margins of the hernial defect. Some of this is undoubtedly due to the aggregation of the displaced fascial fibers about the neck of the hernial sac. In any event, in the presence of hernia, the transversalis fascia frequently assumes a character such that it may be easily sutured with assurance that the repair will hold.

2. *Lateral Border of the Sheath of Rectus Abdominis and the Conjoined Tendon.* In the lower part of the abdomen, the sheath of the rectus muscle is formed by fusion of the aponeurosis of transversus abdominis and internal oblique muscle at the lateral border of the rectus muscle. The aponeurosis of external oblique is also present anterior to the rectus, but in the groin region does not actually fuse with rectus sheath until it nearly reaches the midline; therefore, internal oblique muscle makes no contribution to the lateral border of the rectus sheath which is utilized in hernial repair of the groin. In the common situation, the fibers derived from internal oblique muscle tend to be directed in a more nearly horizontal plane and to insert into the rectus sheath somewhat more superiorly; fibers from transversus abdominis tend to curve inferiorly and medially, to insert along with the tendon of the rectus into the crest of the pubis.

As the aponeurotic fibers of transversus abdominis turn inferiorly contributing to the anterior rectus sheath, they sometimes form a flat band of fibers, a few millimeters to a centimeter in width, just medial to the line of fusion of the aponeurosis and parallel with the border of the rectus. When this band is present it nearly always receives fibers also from the internal oblique aponeurosis.

We have called such a fused aponeurotic band, clearly lateral to edge of rectus, a conjoined tendon. This structure was noted in our dissections to be present about one-third of the time. Griffith [5] and others put the figure much lower, and such differences are probably a matter of definition. What conjoined tendon really represents, it is obvious, is a laterally expanded border of rectus sheath. When the line of transversus-internus fusion is close to the lateral border of rectus muscle, no appreciable band will be present; when the fusion occurs more laterally, then the band will be appreciable and given the separate designation of conjoined tendon. Since the designation of "conjoined tendon" depends on a subjective appreciation of the expanded border of rectus sheath, differences of opinion regarding it are bound to occur.

3. *Henle's Ligament.* This is a small but frequently noted group of fibers often firmly fused with the transversalis fascia, and directed downward and laterally from the lowest portion of the lateral border of the rectus abdominis sheath to insert into the medial portion of Cooper's ligament. This structure is more prominent within the transversalis fascia layer when that fascia is viewed from the preperitoneal side. Henle's ligament is of little importance in groin herniorrhaphy.

4. *Triangular Ligament* (*Reflected Inguinal Ligament*). The fibers of this structure parallel those of Henle's ligament but are located on the anterior surface of the rectus sheath and transversalis fascia, inserting into the pubic tubercle in common with the inguinal ligament, and are not visualized in the preperitoneal approach to the groin. The triangular ligament is of no importance in herniorrhaphy.

5. *Iliopectineal Ligament.* Laterally in the groin where transversus fascia becomes reflected onto the iliacus and psoas muscles there is a thickening of the fascia to form a ligament. This fascial band begins at the anterosuperior iliac spine laterally; the most inferior fibers of the inguinal ligament are attached to its most lateral portion. Arching medially along the surface of iliacus and psoas muscles, it provides origin for the cremaster muscle and the intermingled origins of the most inferior fibers of the internus and transversus muscles. Somewhat

more medially and inferiorly, it gives origin to the iliopubic tract and comes into relation on its medial aspect with the lateral crus of the internal inguinal ring. Continuing inferiorly it contributes to the lateral aspect of the femoral sheath, and terminates by inserting into the pubic bone in the region of the iliopectineal eminence. The importance of this ligament is principally as an anatomic landmark.

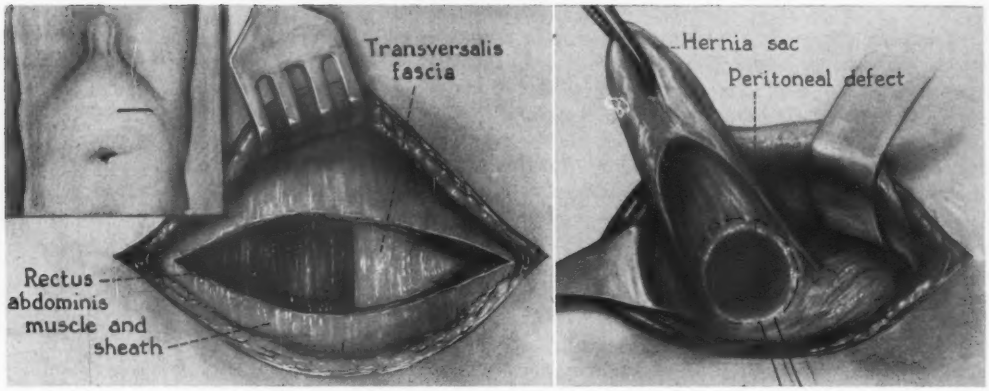
6. Iliopubic Tract. This strong fascial band was easily discernible in three-fourths of our dissections. It originates laterally from the mid-portion of the iliopectineal ligament. It lies immediately subjacent, internal and cephalad to the inguinal ligament in its mid-portion. It is, however, easily separated from the inguinal ligament and its relationship is one of proximity only. From its origin, it arches over the femoral sheath, firmly attached to the anterior leaf of that sheath and turns caudally to insert fanwise into Cooper's ligament throughout its mid-portion. This is the ligament which forms the medial border of the femoral canal, not the lacunar ligament of classic description. In addition to its broad insertion into Cooper's ligament, occasionally a few fibers from this ligament have been noted to travel medially as far as the rectus sheath being located parallel to and about ½ cm. above Cooper's ligament. The importance of the iliopubic tract in hernial repair of the groin can be best appreciated when the posterior inguinal wall is visualized via the preperitoneal approach. It is then readily noted to be a tough, resilient ligament through which the "transitional" or "fatal angle" sutures on the medial aspect of the femoral sheath are placed during the standard repair of an extensive inguinal hernia. The iliopubic tract is regularly employed in the preperitoneal repair of a femoral hernia; when approximated to Cooper's ligament it obliterates the ostium of the femoral canal. It is also regularly employed in preperitoneal repair of an indirect inguinal hernia, when it is used in preference to the thinner more laterally placed crural fibers of the internal ring for anchoring the sutures which close the ring.

7. Crura of the Internal (Abdominal) Inguinal Ring and the Interfoveolar Ligament. The internal ring is ovoid and at its inferior, lateral and medial borders a condensation of tissue is present within the transversalis fascia forming a sling which is open laterally and superiorly. On the lateral aspect, the fibers of the lateral crus spread fanwise to be quickly lost within the general transversalis fascia. The medial crus, in contrast, is a particularly dense, tough band, located immediately subjacent to the origin and proximal course of the inferior epigastric vessels; the vas deferens turns over this band at an acute angle. Occasionally, fibers from the medial crus continue superiorly as a definite band, known as the interfoveolar ligament, as far as the border of the rectus. The medial crus of the internal ring is approximated to the iliopubic tract for performance of repair of an indirect hernia.

8. Femoral Sheath. This is a tubular fascial structure, wider above and tapering below to closely approximate the adventitia of its contained vessels. The ostium of the femoral sheath is continuous with transversalis fascia. It is subdivided by strands of areolar tissue into three compartments. The lateral compartment transmits the femoral artery, and the middle compartment the femoral vein. The medial compartment is the femoral canal, which normally contains nothing but areolar tissue; in the presence of femoral hernia, it transmits the peritoneal sac. In addition to its relationship with transversalis fascia, the femoral ostium is reinforced superiorly and medially by the arching fibers of the iliopubic tract, inferiorly it is in close relationship to Cooper's ligament, and laterally to the iliopectineal ligament. These ligaments are all intimately bound to the uppermost fibers of the sheath providing strong reinforcement. About 1 cm. distal to its ostium the femoral sheath comes into relationship anteriorly with the inguinal ligament which is easily separated from the sheath.

TECHNIC

The preperitoneal area is approached through a lower abdominal transverse incision approximately 3 cm. above the inguinal ligament. Thus this incision is placed only slightly cephalad to that of the standard approach. The rectus sheath is incised transversely at the level of the skin incision, the incision being carried past the lateral border of the rectus abdominis muscle to incise the external oblique, internal oblique and transversus abdominis aponeurosis for a variable distance, usually 4 cm. (Fig. 2A). The rectus muscle is retracted medially, the thin transversalis fascia in front of the preperitoneal fat is incised and the preperitoneal space is entered. The innervation to the rectus muscle is



2A 2B

FIG. 2. A, skin and fascia incision for approach to right preperitoneal area. B, mobilized peritoneal hernial sac with high ligation of the neck of the sac.

not disturbed. By means of blunt dissection, the pelvic peritoneum is readily reflected from the pelvic floor and the peritoneal projections through the pelvic or inguinal floor are easily visualized. Femoral and direct hernial sacs usually can be reduced with minimal blunt dissection. The incarcerated femoral sac may be released by incising the insertion of iliopubic tract into Cooper's ligament at the medial side of the femoral ring. Previous operative descriptions for release of the femoral sac indicated that this incision was into lacunar ligament. Large indirect (scrotal) peritoneal projections may at times be difficult to remove without causing excessive trauma. In this situation, the neck of the sac may be severed, leaving the distal peritoneal sac *in situ*. This latter circumstance is unusual.

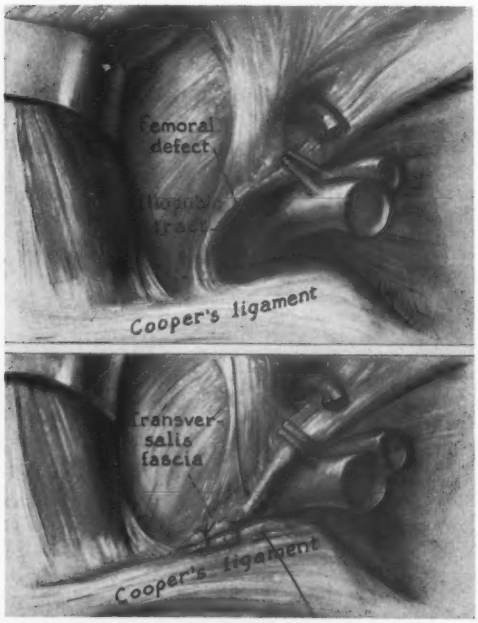
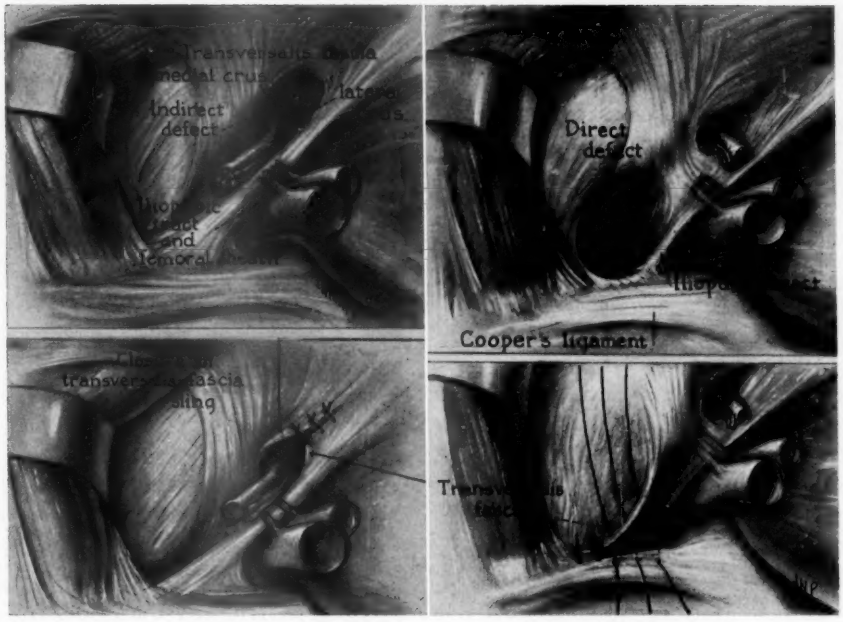


FIG. 3. Anatomy of femoral hernial defect and transversalis fascia repair.

The hernial sac upon mobilization may be invaginated into a purse-string suture or excised with closure of the peritoneum flush with its normal curve. A true high ligation is the rule. (Fig. 2B.) Proximity of the bladder, particularly in direct hernias, must always be considered. The inferior deep epigastric vessels are readily seen in the wound. For complete freedom of movement and better visualization for repair of the fascial structures, it may be necessary to ligate and sever these vessels. When one large hernia is found, there is a tendency to desist from further exploration. It is imperative to visualize carefully each possible site of hernia prior to closure of the wound. Dual and even triple hernias on one side were not infrequent in our experience.

The incision described previously may be extended across the midline in the patient with a bilateral hernia. Visualization is improved and thus ease of repair is greatly enhanced by this approach. We have virtually stopped using the classic vertical midline incision for bilateral hernia.



4 5

FIG. 4. Anatomy of indirect inguinal hernia and repair of transversalis fascia sling.

FIG. 5. Anatomy of direct inguinal hernia and transversalis fascia repair.

In the following description of specific repairs, it should be noted that in every instance transversalis fascia is sutured to itself or an analogue.

Femoral Repair. The superior edge of the defect is either transversalis fascia thickened by "bunching" of the fibers at that point or at that portion of transversalis fascia known as the iliopubic tract. (Fig. 3.) Two or three sutures between the superior margin of the defect, in the iliopubic tract, and Cooper's ligament neatly closes the defect. The external iliac vein is easily seen and protected. The suture material used in this and all repairs is No. 1 black silk.

Indirect Repair. Following high ligation of the hernial sac, traction is placed on the spermatic cord in a medial direction. (Fig. 4.) This delineates the medial crus of the transversalis fascia sling. Continuing the medial traction, the cord is moved slightly superior, and attenuated fibers at the cranial edge of the lateral crus of the sling appear. These attenuated fibers are not used in the repair and must be cut until the more caudal fibers of the iliopubic tract and femoral sheath are seen. These fibers represent the main tissue of the lateral crus. The femoral sheath and iliopubic tract at this level are near but definitely distinct from the inguinal ligament, and for practical purposes are considered to be the true lateral crus of the internal abdominal ring. Sutures are then placed lateral to the cord between the medial crus of the internal abdominal ring (transversalis fascia analogues) and the lateral crus of the internal abdominal ring (iliopubic tract and femoral sheath-transversalis fascia analogues). Three or four sutures usually suffice for snug closure against the cord. McVay [*10*] suggested that in the presence of a massive indirect hernia, complete medial transposition of the cord with lateral suturing would place the closure about the cord in direct line with the external inguinal ring. This certainly is a potentially dangerous situation, and would suggest that in this type defect the medial portion of the repair should be performed as in a direct hernia, followed by lateral closure as indicated above, leaving the cord in the mid-portion of the repair. The repair on the medial aspect of this large defect will necessitate suturing transversalis fascia or conjoined tendon to Cooper's ligament.

Direct Repair. During the initial removal of

TABLE I
DISTRIBUTION OF 213 HERNIAS

Type	Unilateral			Bilateral
	Right	Left	Total	
Femoral	21	15	36	6
Indirect	46	23	69	18
Direct	21	9	30	15
Total	88	47	135	78
			213	

TABLE II
PREVIOUS INGUINAL REPAIR WITH RECURRENCE

Type	Side	Number
Femoral	Left	0
	Right	1
Indirect	Left	6
	Right	11
Direct	Left	4
	Right	11
Total		33

the preperitoneal tissues from the pelvic floor, the tissues in the region of Hesselbach's triangle should be carefully watched. (Fig. 5.) In a direct hernia, the attenuated transversalis fascia at the apex of the peritoneal sac will evert along with the sac. The attenuated fascia can then be grasped with forceps and the margins of the direct defect rapidly demarcated. The inferior margin of the direct defect in many instances is iliopubic tract, and sutures between the thickened transversalis fascia at the superior margin of the defect and iliopubic tract will be adequate for closure. If the iliopubic tract is not a well defined structure the inferior sutures are placed in Cooper's ligament. If the transversalis fascia at the superior margin is of poor quality, a rare finding, the conjoined tendon is found along the superior edge of the defect on the anterior surface of transversalis fascia, and may be brought down to Cooper's ligament. Relaxing incisions, so necessary in the standard hernial repairs, are not necessary in the preperitoneal approach.

Closure of the Wound. Meticulous care must be taken for adequate hemostasis prior to closure of the wound. The preperitoneal space is well irrigated with saline. The thin layer of transversalis fascia entered below the semicircular line of Douglas is carefully closed with medium fine silk. The rectus sheath is sutured with interrupted No 3–0 silk or No. 30 stainless steel wire, followed by closure of Scarpa's fascia and skin.

MATERIAL AND RESULTS

One hundred fifty patients were operated upon between November 1955 and December 1959. This represents a total of 213 separate hernial repairs. (Table 1.) Thirty-three hernias had been repaired previously by a standard

TABLE III
OPERATIVE DEATHS OF PREPERITONEAL HERNIORRHAPHY

Hospital Number	Age, Sex	Type of Hernia	Incarceration or Strangulation	Surgery Other Than Hernial Repair	Cause of Death	Autopsy
177,992	91, F	Right, femoral	Strangulation	Resection of ileum	Peritonitis	Yes
309,880	93, F	Right, femoral	Strangulation	Resection of ileum	Atelectasis, congestive heart failure	Yes
367,637	70, M	Right, recurrent, indirect	Strangulation	Resection of ileum	Pneumonia	Yes
244,732	68, M	Right, indirect	Incarceration	...	Myocardial infarction	No
27,171	67, M	Right, indirect (massive)	...	...	Atelectasis, myocardial infarction	Yes
377,747	73, M	Left, indirect, large, recurrent	...	...	Myocardial infarction	No

TABLE IV
RECURRENCE OF HERNIA AFTER PREPERITONEAL HERNIORRHAPHY

Hospital Number	Age, Sex	Type of Hernia Treated	Time Before Recurrence (mo.)
273,616	47, M	Right, indirect, recurrent	1
341,489	75, M	Right, indirect, recurrent	7
249,081	47, M	Right, indirect, massive	1
345,928	78, M	Right, direct	3
361,266	61, M	Left, indirect	18

type herniorrhaphy. (Table II.) Twenty-six patients presented with incarceration; nine patients presented with strangulation which necessitated resection of bowel. There were six postoperative deaths in the series, four of which followed emergency operations for incarceration or strangulation and all of which occurred in patients with unilateral repair. (Table III.) Postoperative complications included the following: pneumonia, three; genitourinary infection, three; minor wound infection, three; atelectasis, two; and congestive heart failure, two. Five recurrent hernias were found in the follow-up clinic. (Table IV.)

COMMENTS

The history of preperitoneal herniorrhaphy has slowly evolved since its inception as an operative technic in 1920. A review of technics presented for repair of hernias of the groin by this approach clearly shows a gradual evolution of concepts. (Table V.) The ultimate worth of these technics is yet to be proved. We continue to believe that consideration should be given to a statement in our previous paper on this subject that, "the sporadic interest shown in the preperitoneal repair of groin hernias makes one dubious of its ultimate worth. Furthermore, long-term controlled follow-ups are not present in the literature. We plan to continue this study in an endeavor to settle the value of this procedure" [16]. However, our results to date have been encouraging and we are obtaining a new concept of the inguinofemoral anatomy having viewed it from within. Certainly, the dual role of the transversalis fascia in pathogenesis as well as in the repair of

TABLE V
METHODS OF ANATOMIC REPAIR OF DEFECT

Author	Year	Type of Hernia	Method
Cheatle	1920	Indirect	Occlusion of internal ring by suture
		Femoral	Flap periosteum of pubis to Poupart's ligament
Cheatle	1921	Indirect	High ligation of sac only
		Femoral	Flap periosteum of pubis to Poupart's ligament
			Plug femoral opening with coiled autogenous vein
Henry	1936	Indirect	Plastic to internal ring-transversalis fascia to fascia deep surface internal oblique muscle
		Femoral	Flap of pectineus fascia to Poupart's ligament
Jennings, Anson and Wright	1942	Indirect	Plastic closure of internal ring-suture of transversalis fascia sling
Musgrove and McCready	1949	Femoral	Suture of Poupart's ligament to Cooper's ligament
McEvedy	1950	Femoral	Suture of conjoined tendon to Cooper's ligament
Hull and Ganey	1953	Femoral	Suture of Poupart's ligament to Cooper's ligament or pectineal fascia flap technic of Henry
Mikkelsen and Berne	1954	Femoral	Suture of transversalis fascia and transversus aponeurosis to Cooper's ligament
		Small indirect	Plastic repair of internal ring-transversalis fascia
		Large indirect	Similar to femoral closure
Mouzos and Diggory	1956	Femoral	Suture of conjoined tendon to Cooper's ligament
Nyhus, et al.	1959	Indirect	Suture of transversalis fascia sling medial to the cord
		Direct	Suture of transversalis fascia and/or conjoined tendon to Cooper's ligament.
		Femoral	Suture of transversalis fascia and/or conjoined tendon to Cooper's ligament
Nyhus, et al.	1960	Indirect	Suture of transversalis fascia sling lateral to the cord
		Direct	Suture of transversalis fascia and/or conjoined tendon to Cooper's ligament
		Femoral	Suture of transversalis fascia and/or conjoined tendon to Cooper's ligament

hernias of the groin is being delineated. This clarification is of import, regardless of the type herniorrhaphy utilized.

The incidence of recurrent hernia reported herein is too high, approximately 2 per cent of those patients surviving the procedure. All but one of the recurrences followed a repair of an indirect hernia. However, the indirect hernias which recurred were all repaired during the early phase of this study, repaired in a significantly different manner from the technic now used. There have been no recurrences of any type in patients operated upon in the past two years. Specifically, all the indirect recur-

rences were in patients treated by the method published in 1959 [16] (reported in 1957) and none by the method reported in the present paper.

The use of the lower abdominal transverse incision with medial mobilization of the rectus muscle has made this approach exceptionally practical. Whereas, we were loathe to do a unilateral herniorrhaphy by the lower abdominal vertical midline incision, we now believe that all hernias of the groin in adults, whether unilateral or bilateral, may be treated by this technic. We have not used this method for hernias in infants.

Our initial concept that this procedure would be ideal for recurrent hernias previously repaired by the external approach has proved correct to date. The difficulties so inherent in dissecting through the old scar are bypassed. We have operated upon several patients who have had as many as four prior external hernial repairs and found most satisfactory tissue for preperitoneal repair.

SUMMARY

1. The principle of preperitoneal laminar restoration of the anatomy and the importance of the transversalis fascia lamina in hernial repair of the groin are emphasized.

2. The structures which are actually used for repair are a group of local ligaments within the transversalis fascia. We have designated these structures as transversalis fascia analogues. In direct hernial repair, for example, superomedial transversalis fascia is joined to inferolateral transversalis fascia analogues (Cooper's ligament and iliopubic tract).

3. The surgical anatomy of the transversalis fascia and the transversalis fascia analogues is described.

4. One hundred fifty patients with hernias of the groin have been operated upon by the preperitoneal approach; this represents an experience of 213 separate hernial repairs (forty-eight femoral, sixty direct, and 105 indirect inguinal hernias).

5. A recurrent hernia rate of approximately 2 per cent was found. All but one recurrence followed repair of indirect inguinal hernias by a technic discarded by us over two years ago. There have been no recurrences following indirect hernial repair by the technic herein described.

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DISCUSSION

William P. Mikkelsen (Los Angeles, Calif.): The authors deserve signal credit for their continuing investigation into the extra or preperitoneal approach for hernioplasty. Whereas several others have recognized and described the advantages of this approach for femoral hernioplasty, few have recognized its merit for inguinal hernioplasty. I must acknowledge my own inclusion in the latter group. The lucid anatomic descriptions and dia-

grams which the authors present may well serve as a stimulus for the greater consideration of this technic. However, it can not be anticipated that such a radical departure from the tried and reasonably true standard operative approach will be quickly accepted. As is usual in such situations the vast bulk of surgeons will wait for longer follow-up studies. Even if such studies prove the superiority of this procedure most will probably be reluctant to abandon the method with which they are familiar and have had a satisfactory experience. My personal experience in encouraging the greater adoption of the preperitoneal approach for the management of femoral hernia has been most successful with the surgeons in training. These men are adaptable. With this in mind, it behooves the teachers of surgery to examine critically this method of hernial repair; if found worthy, it should be included in their program of surgical instruction.

In addition to the management of femoral hernias, the preperitoneal approach is appealing for the management of sliding hernias. The hazard of inadvertent penetration of the bowel and the necessity of the La Roque extension procedure frequently encountered in the standard approach would appear to be largely avoided.

In contrast one wonders how much difficulty is encountered in the release of a strangulated indirect inguinal hernia, the locking mechanism of which is usually the external inguinal ring and which lies superfical to the plane of preperitoneal dissection.

The authors' experience with postoperative recurrence has been predominantly with indirect inguinal hernias. It is of interest that their current method of repairing this hernia embodies the principles laid down eighteen years ago by Kenneth Jennings, a member of this association. This involves lateral rather than medial repair of the internal inguinal aperture; the cord structures are thus displaced to the medial rather than the lateral boundary of this aperture. This experience, perhaps should stimulate an inquiry into the justification for the lateral displacement of the cord structures when the external or standard operative approach is used.

Although a relaxing incision, so commonly employed in the management of large inguinal hernias by the standard approach, was not used by the authors in their present series, it would seem that there might, nevertheless, be instances when it would be advantageous. With this in mind, I recently dissected a few fresh cadavers and was pleased to note that such a relaxing incision could be quickly and easily accomplished through the preperitoneal approach.

Finally, I should like to speak for the midline vertical or Pfannenstiel incision for femoral hernias at least. In this preperitoneal technic the rectus muscles are displaced laterally rather than medially as the authors describe. Ample exposure is thus obtained and the opposite hernial aperture can be rapidly exposed and repaired with little added dissection and no added incisions, either in the skin, fascias or muscles.

W. Kenneth Jennings (Santa Barbara, Calif.): Both Drs. Nyhus and Mikkelsen have referred to my interest in this subject. It began some twenty years ago when I read Professor Henry's article. We had used his proposed technic in the repair of several femoral hernias and were quite impressed with the exposure of the opening of the femoral canal which this retroperitoneal approach provided. We were also interested to observe that a little additional lateral retraction of the rectus muscle resulted in excellent exposure of the abdominal inguinal ring.

It occurred to me that this would offer a simple means of insuring a high ligation of the indirect hernial sac which, together with the natural obliquity of the inguinal canal and the placement of two or three sutures in the transversalis fascia, would effectively close the abdominal inguinal ring and thus afford adequate protection against recurrence.

I suggested this idea to Dr. Barry Anson, who then was Associate Professor of Anatomy at Northwestern University and who, together with Chester McVay, was very much interested in the anatomy of the inguinal area, particularly as it related to hernias of the groin. He proposed performing a series of dissections of cadavers reversing the usual procedure of beginning with the outermost layers of the inguinal wall and rather starting the dissection from the intraperitoneal side exposing each layer as encountered when progressing from within outward.

Dissection was performed through the acetabula and the ischial rami and a coronal section through the abdomen was carried down through the pelvis.

On the right side, the peritoneum was intact, and a congenital hernial sac was noted. On the left side the peritoneum was removed, exposing the preperitoneal connective tissue, which likewise was removed over the epigastric vessels and the umbilical ligament to show their relationship to the abdominal inguinal ring.

The transversalis fascia, in the instance of hernia, often enters the canal to participate in the investment of the spermatic cord. The relationship of the epigastric and external iliac vessels to the abdominal ring was seen in this dissection.

Abdominal and femoral inguinal openings were seen with wide lateral retraction of the rectus muscle, as suggested by Dr. Mikkelsen. The spermatic vessels converged to form the cord vessels, ductus deferens and transversalis fascia on the lateral margin. This shows how easy it is to

effectively close the femoral opening simply by suturing the iliopubic ligament or, in this instance, the inguinal ligament to the Cooper's ligament.

In the article which we published in *Surgery, Gynecology and Obstetrics*, in 1942, we proposed the following technic. A low vertical incision is made and carried down to the peritoneum, which is then separated from the rectus muscle by blunt dissection. The rectus muscle is retracted laterally, exposing the abdominal inguinal ring. The congenital hernial sac is separated from the cord structures, opened and high ligation is accomplished. Two sutures are placed in the transversalis fascia to close effectively the abdominal ring. Then there is nothing left to do except to close the midline incision.

For the next several years I was quite enthusiastic about the operation, and in 1942 published in *Surgery, Gynecology and Obstetrics* our experiences with its use. To quote from our conclusions: "In the restoration of an adequate parietal barricade to hernia, the serous sac is, of course, the fundamental anatomical element; its space is the only true inguinal canal. Since its congenital presence offered the initial route for hernial progress, its obliteration logically consitutes the primary step in surgery of repair. Furthermore, in carrying out the ligation of the peritoneal sac, that method would seem most rational which brings minimal distortion and stretching to overlying parietal layers which have already been inordinately stretched."

However, with continuing experience, we believed the operation had certain limitations. In the long-standing, large, indirect inguinal hernia the abdominal and subcutaneous inguinal openings are so stretched that one underlies the other, creating in effect a direct hernial opening; it seems that in private practice most of the inguinal hernias examined were of this variety. We were afraid to depend upon a simple transversalis fascia repair for such weaknesses and came to favor the conventional approach together with McVay's relaxing incisions in the anterior rectus sheath, suturing the lateral fascial margin to Cooper's ligament.

I still believe that for small congenital hernia the retroperitoneal technic provides an ideal anatomic repair, and if offers the advantages of (1) preserving the anterior abdominal wall in the groin area intact, (2) less chance of wound infection from silk or cotton fistula formation, (3) a reduced likelihood of femoral phlebothrombosis because of less pain and, finally (4) it makes possible a repair of bilateral inguinal hernia through a single incision, whether it be the vertical incision of Professor Henry of Cairo, Egypt, or the transverse incision proposed by Dr. Nyhus.

LLOYD M. NYHUS (closing): As to the problem of the incarcerated indirect hernia, which might be incarcerated at the external ring, it is simple to perform a relaxing incision by lifting up the skin and to dissect subcutaneously down to the external ring. It would be quite simple to handle. We have not had any trouble in that regard.

I think it is most important, if you use the Jennings closure for the large ring, to perform an additional repair of the Cooper ligament type on the medial side of the sling repair.

I should like to re-emphasize two more things. We believe quite strongly that the lower abdominal transverse incision has made it the most practical procedure, whereas before we were loathe to make the vertical midline incision for unilateral hernia. We now believe that the transverse approach can be utilized for all hernias of the groin in adult patients, whether the hernia is unilateral or bilateral.

Our initial concept that this procedure might be ideal for recurrent hernias previously repaired by an external approach has proved correct. The difficulties so inherent in dissecting through an old scar are obviated. We have operated upon several patients who have undergone as many as four prior herniorrhaphies performed by the standard approach and have found most excellent tissue for preperitoneal repair.

Vagotomy and Pyloroplasty for Acute Perforated Duodenal Ulcer

A REPORT OF SEVENTY-FIVE CASES

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SIMPLE operative closure is the most widely accepted form of treatment for acute perforated duodenal ulcer. The chief virtue of simple operative closure is that the patient is subjected to a minimum of operative stress and trauma when he is seriously ill. Long term follow-up studies have shown that simple closure alone has no basic definitive effect on the ulcer diathesis. Many studies indicate a 60 to 80 per cent recurrence rate of ulcers in patients having undergone simple closure. Also, it has been shown that up to 60 per cent of patients with simple closure may ultimately require definitive surgical treatment [*1,9,10, 12–14*]. The majority of patients who undergo simple closure with poor long term results probably are those who have well established chronic duodenal ulcer disease. Gastric resection has been performed in such patients with minimal mortality and morbidity [*2,3,5,6,12, 15,16*]. Nevertheless, many object to the use of a procedure of such magnitude as gastric resection in these seriously ill patients. Also, with primary gastric resection, some patients are unnecessarily subjected to undesirable postgastrectomy sequelae.

Vagotomy with pyloroplasty or gastroenterostomy is an acceptable definitive operative procedure for duodenal ulcer. The operative mortality rate attending vagotomy and pyloroplasty is usually considered lower than that of gastric resection. In addition, the postoperative sequelae, such as the "dumping syndrome" and nutritional deficiencies of various types, are less prominent after vagotomy and pyloroplasty than after the average gastric resection. Vagotomy with pyloroplasty or gastroenterostomy is a definitive surgical procedure for duodenal ulcer which can be applied to cases of acute duodenal perforation. The authors have previously reported thirty-six selected cases of acute perforated duodenal ulcer treated by vagotomy and pyloroplasty without mortality and minimal morbidity [*11*]. This is a subsequent report based on the observation of seventy-five patients treated by this method.

METHOD OF SELECTION OF PATIENTS

The criteria for selection of patients, as previously reported, are as follows: (1) no patients over fifty-five years of age were included; (2) perforations were relatively early, usually less than twelve hours; (3) patients with excessive peritoneal spill were excluded (young favorable patients with larger spills were sometimes included); (4) no patients with persistent shock were used; and (5) patients with severe systemic disease or acute alcoholism were not included [*11*]. In this series, over 90 per cent of patients had a history of chronic ulcer disease.

OBSERVATIONS ON OPERATIVE TECHNIC

Upper midline incisions are used, and if the patient's tolerance to anesthesia is satisfactory, a Heineke-Mikulicz type pyloroplasty followed by a standard subdiaphragmatic vagotomy is performed. If the duodenum is unsuitable for pyloroplasty, simple closure may be performed with vagotomy and gastrojejunostomy. This was necessary in one of our patients.

The Heineke-Mikulicz type pyloroplasty is readily adaptable to the acute perforated

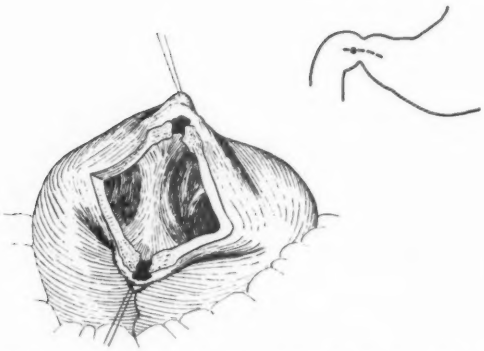

FIG. 1. Transverse closure of longitudinal incision through the typical mid-anterior ulcer and pyloric ring.

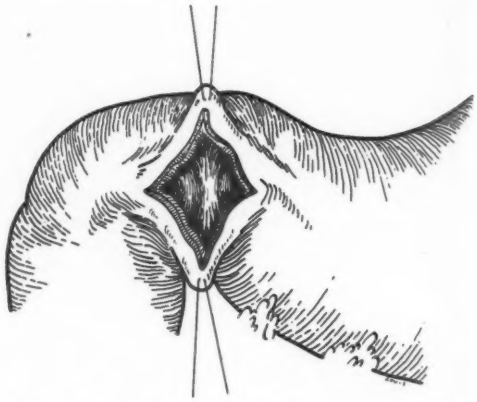

FIG. 2. Diamond-shaped excision of the indurated margin surrounding the ulcer.

duodenal ulcer for several reasons. Most perforations are small in size and are usually located close to the pylorus in a mid-anterior position. Most perforations have less than 1 cm. of severe surrounding induration. The duodenum is usually not fixed to the pancreas and when the lateral peritoneal reflection is incised (Kocher maneuver), excellent mobilization is obtained, facilitating pyloroplasty. When the longitudinal incision through the typical mid-anterior ulcer and pyloric ring is closed transversely (Heineke-Mikulicz), the indurated margins of the ulcer move to the sites of least tension, that is, the superior and inferior poles of the transverse closure. (Fig. 1.) Consequently relatively normal tissue is present in the central part of the transverse closure, which is the point of greatest tension in the pyloroplasty. A diamond-shaped excision of the indurated margin surrounding the ulcer was performed in most of the later cases in this series. (Fig. 2.) The occasional perforations which are located at a distance from the pylorus are the most difficult to manage and may require the use of gastroenterostomy in place of pyloroplasty. The incidence of associated duodenal ulcer of the posterior wall is at least 13 per cent in this study. Direct visualization of these lesions is possible during pyloroplasty. The presence of these ulcers of the posterior wall did not interfere with the pyloroplasty procedure.

The actual transverse closure of the pyloroplasty (Figs. 3 and 4) is accomplished utilizing an inner mucosal row of fine intestinal catgut

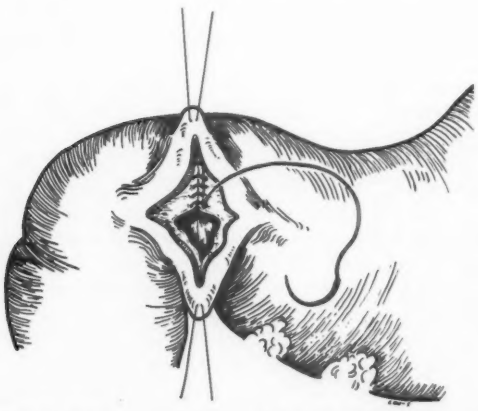

FIG. 3. Inner mucosal closure with a row of fine intestinal catgut suture.

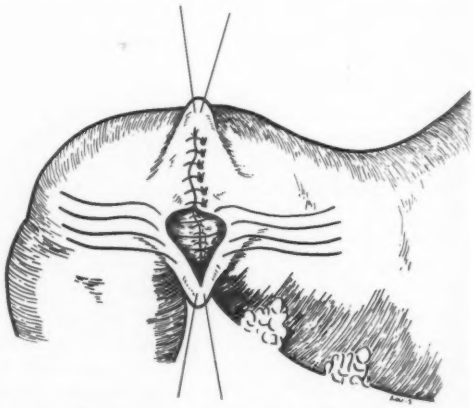

FIG. 4. Seromuscular closure with interrupted silk suture.

suture and interrupted silk sutures for the seromuscular row. Figure 5 illustrates this two-row, one-layer technic which avoids the inversion of a large cuff of tissue with its potential dangers of duodenal obstruction. Nasogastric suction was used for two to four days postoperatively.

RESULTS

There was one postoperative death in this series giving a mortality rate of 1.3 per cent. There were two significant complications resulting in a morbidity rate of 2.6 per cent. The average hospital stay was nine days and the average age of patients was forty-one years. In general, the postoperative course of these patients was smooth and uneventful.

COMMENTS

The chief objections which might be raised to the performance of vagotomy and pyloroplasty in the presence of acute duodenal perforation are mediastinitis and duodenal leakage. Neither of these problems occurred in this group of seventy-five patients. The only serious complications encountered were a pelvic abscess which cleared following surgical drainage and one instance of delayed gastric emptying requiring gastroenterostomy on the twenty-sixth postoperative day. The one death which occurred was on the sixth postoperative day. An autopsy showed secure suture lines and no evidence of mediastinitis of esophageal perforation. In review, it was believed that death resulted from hypovolemia and electrolyte deficiencies.

It seems likely that the sharp decrease in secretion of gastric juice resulting from vagotomy and the effective gastric drainage from pyloroplasty favorably influenced the postoperative course of these patients. A non-obstructive, well sutured pyloroplasty closure seems surgically secure when compared with some simple closure technics in which severely indurated margins are approximated. Vagotomy with pyloroplasty or gastroenterostomy is an operation which requires more surgical experience than does simple closure. However, in the hands of those accustomed to the procedure, it appears to be a definitive operation which can be performed with minimum mortality and morbidity.

It should be emphasized that this is still a preliminary study and no final conclusions regarding long term results can be made at this time.

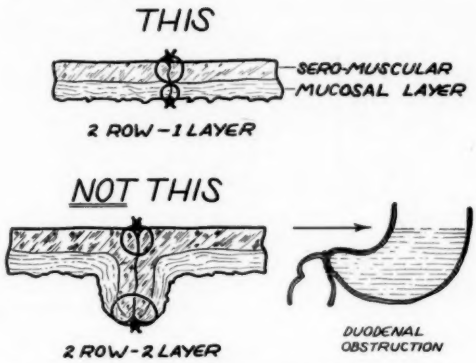

FIG. 5. Cross-section demonstrates the two-row, one-layer technic of suturing.

Long term follow-up studies are in progress and will be subsequently reported.

SUMMARY AND CONCLUSIONS

1. Later definitive surgery is frequently necessary when simple closure has been used in treating a patient with perforated duodenal ulcer.

2. Vagotomy with pyloroplasty or gastroenterostomy is a definitive operative procedure for duodenal ulcer of lesser magnitude than gastric resection and it is usually associated with less mortality and fewer postoperative sequelae. Vagotomy and pyloroplasty has been shown to be technically adaptable to the surgical treatment of acute perforated duodenal ulcer.

3. It is interesting to note the associated incidence of posterior duodenal ulcer in these patients with anterior duodenal ulcer perforation.

4. Vagotomy with pyloroplasty has been successfully used in the treatment of seventy-five patients with acute perforated ulcer, with a 1.3 per cent mortality and with minimal morbidity.

5. This study is still in a preliminary stage and long term follow-up observations will be subsequently reported.

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DISCUSSION

Jack M. Farris (Los Angeles, Calif.): Those of us who are following this particular field are aware of the importance of the report previously presented. A series of seventy-five patients with perforated ulcers treated definitively is certainly a unique one.

It certainly is clear that definitive surgery or operation may now be safely performed in the presence of perforation. Most physicians have been extremely disappointed with simple closure of a perforated ulcer because shortly after convalescence, the patient returns to his ulcerogenic environment and we have accomplished little as far as his fundamental problem is concerned.

We also agree that definitive operation should only be performed by those who have had experience and are particularly skilled in this field. It certainly behooves us not to teach the definitive operation as to be universally employed by one who operates on ulcers occasionally. The good results previously presented are due in large part to, first of all, the patient considered a superior surgical risk (average age of forty-one years); secondly, a short interval between perforation and operation; and thirdly, the experience these men have had with this particular operation.

Concerning ulcers in general, the fact that an ulcer perforates does not alter the fundamental problem, nor does the fact that an ulcer bleeds, for example, alter the problem of the ulcer diathesis.

I would like to make a few remarks about the ulcer diathesis in general. If we agree that definitive surgery may be performed in certain instances safely, do we agree upon what is the proper or acceptable operation in the surgical treatment of duodenal ulcer? What is the efficacy of pyloroplasty and vagotomy compared with other types generally employed in the United States today?

The physiologic information we have at the present time emphasizes that the vagus apparatus is becoming more and more important in the ulcerogenic mechanism, and perhaps the antral phase (or the pyloric gland area phase) in duodenal ulcer, at least, is becoming less and less important. It is now clear that stimulation of the vagi releases gastrin, to support the Edkins hypothesis. It is also quite clear, I think, that acid in the antrum produces an inhibitory effect upon the fundus or corpus, and parietal activity. Also the hypothesis that the pyloric gland area inhibits ulcer formation in animals is pretty well supported at the present time. In addition, pyloroplasty (properly performed) is just as effective as antrectomy in nullifying this phase of the mechanism.

We have been carrying out some studies in an effort to increase the scope of the vagotomy operation. Most important information has been achieved or obtained from the mounting of a normal esophagus in a paraffin block, making a cross section *in toto* and examining the periphery of the esophagus for nerve fibers. In a normal esophagus the anterior nerve may have nine or ten trunks, the posterior nerve may have almost as many, and sometimes one may count anywhere from twenty to thirty nerve trunks at the periphery of the esophagus.

One of my patients died on the third postoperative day following vagotomy and pyloroplasty from a coronary occlusion. On viewing the esophagus, two, rather large, nerve trunks were noted. I am not certain that the finding of these residual nerve

trunks will nullify the effects of vagotomy, but I am certain that it is important for us to make more careful searches for residual nerves.

A specimen of vagus nerves was removed at the operating table. This fiber was almost 7 or 8 cm. in length, being the posterior one. The numerous branches and the double nerve trunk frequently found anteriorly were also noted.

Clips were placed on the proximal ends of these nerves at the time of division. A postoperative roentgenogram of the chest showed the clips to be well within the mediastinum. We removed exactly the same fibers that we used to by transthoracic operation.

A word about the efficacy of pyloroplasty. We, for the past year, have performed pyloroplasty by simple seromuscular sutures, omitting sutures in the mucosa at all. This seems to give better results and does not leave non-absorbable sutures in the lumen.

One of my patients had an enormous duodenal ulcer. It measured 2 inches in diameter. It also had a large blood clot in the center of it. The patient had a hemoglobin of 5 gm. He actually fainted because of cerebral anoxia. This was the largest ulcer I had ever seen; it had destroyed the postduodenal wall.

The patient was subjected to pyloroplasty and vagotomy, and transfixion and ligation of the bleeding vessel, and then, through the temporary gastrostomy tube, a water-soluble radiopaque substance was introduced at twenty-four, forty-eight and seventy-two hours. Forty-eight hours after operation, a diminution of about 50 per cent in the size of the ulcer was noted, and fourteen days later it was impossible to demonstrate the crater definitely.

This emphasizes, I think, that it is not necessary to remove duodenal ulcers, and if one actually solves the diathesis, healing will progress at an amazingly rapid state. Removal of duodenal ulcers is unimportant.

I am certain that as the years go by there will be less and less emphasis upon the quantitative extirpation of gastric mucosa as the solution to a problem which fundamentally is related to inability of man to adapt himself to his environment. There will be more and more emphasis, I think, on more subtle attacks upon the physiologic principles involved, such as the vagus apparatus, and an adoption of operations, such as pyloroplasty, which preserves the stomach and at the same time nullifies the role of the pyloric canal in the antral phase of hypersecretion.

ALFRED STRAUSS (Chicago, Ill.): The Strauss Surgical Group in Chicago has not been in favor of subtotal gastric resection in the presence of perforation. We noticed that when we performed simple closure, which we did up until about 1928, about 80 per cent of the patients returned with an obstructed duodenal ulcer which later necessitated subtotal resection. Therefore, we devised a pyloroplasty in 1928.

We have invented a simple instrument, a metal fishhook type spear, for this procedure to hold the perforation in place. This is placed over the perforation, and forced through the muscular walls of the infiltrated areas. A longitudinal elliptic incision is made through the pyloric ring into the duodenum. The entire area is widely excised and includes the greater portion of the anterior part of the pyloric sphincter, leaving only normal tissue. Then the elliptic incision is sutured transversely with fine catgut and a fine layer of inversion wax silk suture (Heineke-Mikulicz procedure).

In the early days, when good anesthetists were scarce, we performed many of these with the patient under block local anesthesia because many of these patients came to us rather late and in poor condition. Later on, we used intravenous sodium pentothal plus block anesthesia.

The entire procedure does not take more than twenty minutes and is little more than a simple closure. Our reason for performing this is to avoid patients coming back immediately for an obstructed ulcer. Only 28 per cent of 250 patients treated for perforations returned with an obstructed ulcer.

Recently, some of us have added vagotomy to this procedure, and I think it is an excellent thing. We did not know much about vagotomy in 1928, but we believe that this does away with the idea of doing definitive or subtotal resection at the time of perforation, and prevents the necessity for a second operation.

JOSEPH A. WEINBERG (Long Beach, Calif.): This paper by Drs. Pierandozzi, Hinshaw and Stafford must be regarded not only as a great step forward in dealing with the perforated duodenal ulcer, but also as a further recognition and application of the epoch-making work of Dr. Dragstedt. What is going on in our present day in connection with ulcer therapy through the schools of Dragstedt and Harkins will be long remembered as the inception of the physiologic approach to the surgical management of peptic ulcer.

Our group has been aware of the work of Dr. Pierandozzi and his associates, and we have gradually begun to use their method. I will admit that we had some fears at first that there might be enough contamination of the mediastinum at the time of performance of the vagotomy to create a hazard of mediastinal infection. However, there has been no clinical evidence of mediastinitis thus far and our results have been completely satisfactory in this and other respects. The number of operations of this type which we have performed is far less than the number that Dr. Pierandozzi has reported, but

we have had enough experience to convince us that this is an excellent manner of dealing with duodenal perforation which is seen early.

There are a few points which I would like to make concerning technic. One should not close the opening which is made through the esophageal hiatus in performing the vagotomy. This is an objectionable procedure at any time because of the danger of allowing blood or other fluid to accumulate in the mediastinal space, and the hazard would seem to be the greater if contaminated material is allowed to be retained in the mediastinum because of closure of the hiatus. Dr. Pierandozzi did not state whether or not he leaves the hiatal aperture open following vagotomy.

I am opposed to mobilization of the duodenum, the so-called Kocher maneuver, in dealing with perforation. This maneuver necessitates opening of the peritoneum with exposure of subperitoneal tissues. On theoretical grounds at least, this might encourage the formation of subhepatic or subperitoneal abscess. I find it rarely advisable to mobilize the duodenum even in cases of nonperforation and avoid it because of the danger of creating adhesions with its use. I would consider the practice doubly objectionable in the case of perforated ulcer.

It is unnecessary to divide the triangular ligament to free the left lobe of the liver in performing vagotomy. I have not found this to be an aid in exposure of the hiatus. It is actually a handicap, because the left lobe of the liver is not as easily controlled after its ligament is divided. Here again, the contamination incident to perforation makes the practice doubly objectionable. Dr. Pierandozzi has not dealt with this phase of the technic, obviously because of lack of time to consider all details.

I would seriously object to the use of the continuous suture which Dr. Pierandozzi said he sometimes uses in closing the pyloroplasty wound. The continuous suture does not give as accurate approximation of tissues as interrupted sutures would give, in my opinion. There is also the objection that if the suture breaks at any point the entire line of suture is weakened with possibility of leakage. In our own clinic, we have yet to experience clinical evidence of leakage with the use of single row interrupted suturing in now more than 850 operations of pyloroplasty.

I would like to make one more comment. Reference has been made to "average age of forty-one years." There is objection to use of the term "average age" because of its failure to convey definite information. In this instance, if half the patients were sixty years old and half the patients were twenty years old, there would be an average age of about forty years, yet none would be at the actual age of forty. It would be much more informative to give the actual number of patients in each of several decades.

JOHN S. PIERANDOZZI (closing): We agree with Dr. Farris that this procedure should be performed by well trained surgeons, certainly not by the occasional operator, and preferably by those who have experience in performing elective vagotomy-pyloroplasty procedures.

In reply to Dr. Weinberg, we do not close the hiatus area.

As for the technic of pyloroplasty, we do use the interrupted silk seromuscular method of closure. The mucosal area is sometimes closed with a running chromic stitch and sometimes with an interrupted stitch.

Most of our patients were between thirty and fifty-five years of age. I agree that probably a mean age would be preferable to report.

Billroth I Gastric Resection for Chronic Duodenal Ulcer

WILLIAM B. HUTCHINSON, M.D. AND LAWRENCE B. KIRILUK, M.D., *Seattle, Washington*

WHEN the story of the surgical treatment for chronic duodenal ulcer is reviewed it is interesting to note that the cycle from minimal to maximum interference and back again has almost been completed. Gastroenterostomy and pyloroplasty lost popularity because of the frequency of recurrent ulceration. The same criticism has been directed towards the Billroth I resection. The Billroth II resection has the lowest incidence of ulceration but the highest operative mortality rate, and surely the factors of malnutrition and weight loss in some patients are an irrefutable fact. Currently with the introduction of vagotomy and the limited resection or drainage procedure recurrent ulceration is alleged to be reduced to a minimum and the problem of malnutrition and weight loss eliminated. The latter group will be properly evaluated with the passage of time. Many physicians are ready to admit that the completely ideal operation for chronic duodenal ulcer, if this can be considered the answer, probably has not yet been devised. The mere fact that so many different operations are advocated for the treatment of the same problem is good evidence for such a statement. The removal of a sphincter in the body is associated with serious consequences and the pyloric sphincter is no exception. The removal of great portions of the stomach, severance of the vagus nerves, and new connections all alter tremendously the physiology of the stomach causing good or evil effects, depending on how the results are interpreted and the medical mathematician calculating these results. Nevertheless the over-all picture has been good and the patient with a serious problem from chronic duodenal ulcer has been rehabilitated regardless of the operation used in about 85 per cent of cases. These good results may in some measure be due to the readjustments of the patient as a whole and not entirely to the surgical rearrangement of the physiology or anatomy of the gastrointestinal tract. Careful attention to the details of selecting the proper procedure for the proper patient can, we believe, reduce the remaining 15 per cent to less than 3 per cent that would be classified by any physician as a poor result.

This paper has for its purpose the presentation of a group of patients operated upon over the past five years for chronic duodenal ulcer. They were all subjected to a 75 per cent or more gastric resection with a Billroth I type of reanastomosis and represent only a portion of our patients operated upon for chronic duodenal ulcer. Full consideration was given to the individual personality, age, the severity and type of duodenal involvement and the mobility of the structures to be approximated. The final selection of patients subjected to this procedure was done at the operating table. Those with more severe mechanical problems were subjected to other procedures. The type of anastomosis performed was never decided upon until the stomach had been resected. We are absolutely sure that in no instance was 1 cm. less stomach removed in any patient in order to anastomose it to the duodenum. In our patients receiving the Billroth I resection the mechanics of bringing the duodenum and stomach into close proximity for anastomosis were not difficult. Thus, the Billroth I operation was not forced, not featured, and actually was performed in patients who theoretically, we believe, should have had the best clinical results.

It is our intention simply to present the clinical results we have obtained with the Billroth I operation for chronic duodenal ulcer over a short period of time and with a short follow-up period. It is reasonable to expect that

as the follow-up period is extended the number of complications will increase. Our follow-up study has been 100 per cent.

It is also our intention to discuss our ideas in relation to our experiences with the Billroth 1 operation and not to compare this procedure with other operations. We have not included and will not consider operations for gastric ulcer or gastric carcinoma which are different problems. We subjected sixty-one patients to a Billroth 1 resection for chronic duodenal ulcer during a five-year period, from September 1954 to September 1959. There were thirty-eight male and twenty-three female patients. There was only one patient under forty years of age. No operative deaths occurred. The gastroduodenal anastomosis was performed in two ways. In forty patients the anastomosis was between the duodenum and the lesser curvature of the stomach, and in twenty-one patients the duodenum was anastomosed to the greater curvature. During this relatively short follow-up period there have been six recurrences as proved by the clinical symptomatology and positive x-ray findings in each instance. In two of these cases bleeding has occurred of moderate severity. All of the patients with recurrences have responded to medical management, but are not cured and not entirely symptom free. None of the six as yet have required reoperation.

The irony of the situation becomes more evident when one realizes that, as with many other surgical procedures, there is often a lag of time between the surgical procedure and the appearance of the complications which may be attributable to the procedure or the recurrence of the specific entity which the procedure was designed to cure. In our series the recurrence rate is 10 per cent. This is not different from some others reported.

Walters et al. [*12*] report an admittedly small series of twenty-seven cases of Billroth 1 operations performed for duodenal ulcers. They report a 7 per cent recurrence and a 7 per cent suspected recurrence.

Goligher and co-workers [*3*] reported eighty cases of duodenal ulcer treated with the Billroth 1 operation and followed up three or more years. They found eleven (13.7 per cent) recurrences and three (3.7 per cent) suspected recurrences.

Hickenbotham [*6*] reports a recurrence of 11.5 per cent in ninety-five cases of Billroth 1 resections for duodenal ulcer. The average time of recurrence was two years postoperatively.

Harkins et al. [*4*] report a much lower incidence of recurrences in a series of 493 operations performed for benign gastric and duodenal disease.

In view of these varied clinical results obtained in the previously reported series, what factors must be considered? The timesaving advantage is important to both patient and surgeon. The operation is performed in the upper abdominal compartment which lessens the problems of obstruction, fistulas, and the like. The Billroth 1 operation is alleged to be a "physiological anastomosis" with the gastric juices draining into the duodenum. However, the Billroth 1 resection may not be as physiological as it appears. The fact that the duodenal mucosa is more resistant to acid peptic digestion than is the jejunal mucosa is open to question. The experimental work done by Kiriluk and Merendino [*7–9*] reveals that the mucosa of the duodenum and jejunum is similar in sensitivity, but that the incidence of ulceration depends upon the amount of buffering capacity of the alkaline, bile and pancreatic juices present at that site. The experimental evidence indicating that there is inhibition of gastric secretion when acid is present in the duodenum may be a factor (duodenal inhibition), but this must be a minor factor since in the normal situation of a duodenal ulcer there is almost a constant flow of acid and yet there is a hyperacidity in the stomach. The mere presence of a duodenal ulcer is evidence in itself that the duodenal mucosa is unable to cope with hyperacidity. The incidence of "dumping" and so-called "intestinal disturbances" is about the same in comparable types of gastric resections. However, studies [*1,10,14*] reveal that the nutritional status, due to better absorption of fat and nitrogen, is maintained better in Billroth 1 than in some other procedures.

It is interesting to postulate why the recurrence rate is high in the Billroth 1 procedure. We believe that there are two reasons for this occurrence. First, there is a spacial separation of the alkaline bile and pancreatic juice and the acid peptic mixture. Secondly, the pyloric sphincter is removed, allowing the acid peptic mixture to constantly flow over the duodenal mucosa. These of course go hand in hand. The mechanism probably does not hold true in all cases but does occur in a sufficient percentage to

increase the number of recurrent ulcers. Generally, the patient with ulcers has a hyperactive bowel. Thus, with a separation of 6 to 8 cm. of duodenum between the anastomosis line and the ampulla of Vater, regurgitation may not occur in all cases, hence there is no neutralization of acid. In support of this theory one needs only to examine the work of Henley [*5*] who interposed a segment of jejunum between the gastric remnant and the duodenum. In the interposition operation the recurrence rate was reported to be 8 per cent within two years [*11*], and one would presume this has risen to prohibitive levels as evidenced by the lack of published reports. Another example is gastrectomy performed with a Roux–Y anastomosis, wherein the recurrent ulceration rate is up to 50 per cent [2]. A more exaggerated example of spacial separation is the Mann-Williamson preparation. This spacial separation of alkaline juices and acid prevents neutralization and thus enhances duodenal irritation by the constant flow of acid peptic mixture from the stomach. In the normal stiuation the pylorus acts as a valve allowing only intermittent flow.

Regardless of how easy or attractive the theoretical advantages appear to be in any surgical procedure, the real value of the procedure must be judged by its clinical results. The results in our small series of patients have conclusively proved to us that the Billroth I operation for chronic duodenal ulcer is associated with a high rate of recurrent ulcers. This high recurrence rate of ulceration will outweigh the advantages of the Billroth I operation for the cure of chronic duodenal ulcer and should not be used for this reason. It is understandable why Von Haberer admitted failure in 1939 after performing about 2,000 of these operations [*13*].

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DISCUSSION

Henry Harkins (Seattle, Wash.): I would like to comment on first, the general situation regarding the Billroth I gastrectomy for duodenal ulcer, and second, a discussion as to why our recurrence rate following this procedure is apparently better than in some other series reported, including Dr. Hutchinson's.

Regarding the general results, mortality must be considered. There is essential agreement that the Billroth I procedure does not add to mortality. In fact in our hospital, it is followed by a lower mortality than the Billroth II procedure used in comparable cases. Furthermore, I believe practically everyone agrees that the Billroth I does at least as well as the Billroth II procedure with respect to general morbidity, weight disturbance, postgastrectomy anemia and dumping. This has been shown both experimentally and clinically and has been our experience.

In addition, I believe there is almost unanimous agreement that the Billroth I procedure presents many advantages and no disadvantages when applied to resection for gastric ulcer.

Regarding the incidence of recurrence, I will preface my remarks by stating that for some six

years up to September 1, 1955, our preferred procedure for the surgical treatment of duodenal ulcer was a 75 per cent resection using Billroth 1 procedure. At that time we had had experience with this procedure for 250 duodenal ulcers. On September 1, 1955, we switched to a 50 per cent resection, removing all the antrum, plus vagotomy, and gastroduodenal anastomosis. Our reason for switching to this newer procedure which we term the "combined operation" was not because of any bad experiences with the 75 per cent Billroth 1 resection as far as recurrence is concerned, but because of our observation that the Billroth 1 procedure alone had not appreciably lowered the incidence of dumping which had previously led us to abandon the Billroth 11 procedure in 1949. In other words, it was to lower the dumping incidence rather than improve the recurrence rate that we adopted the "combined operation." I will not refer to this newer procedure again except to say that our preliminary observations seem to justify its adoption.

This leads to an explanation as to why some groups, including Dr. Hutchinson's, have reported a higher incidence than we have of recurrence following the 75 per cent Billroth 1 resection without vagotomy. I will list some of the possible explanations.

1. Type of Ulcer. In both Dr. Hutchinson's and our series, the ulcer was duodenal which is of course quite relevant as the incidence of recurrence following any Billroth operation, 1 or 11, is much lower if gastric ulcers are included. Thus, Dr. Norman Tanner of London, who does not use the Billroth 1 procedure for duodenal ulcers, has had only two recurrences in a series of 800 Billroth 1 operations for gastric ulcer (0.25 per cent). In addition, I think that the cases of ulcers encountered at King County Hospital are at least as bad as those reported by Dr. Hutchinson, and Dr. Kiriluk, a graduate of our residency program, will vouch for the fact.

2. Age of the Patients. Our patients are older than those reported in any other large series. They averaged fifty-seven years of age and 72.3 per cent were over fifty years of age. It may be that our patients have atrophy of the stomach due to age and hence are not so prone to recurrence. But at least there is the difference that our patients are older.

3. Criteria of Recurrence. We have had a few patients with mild postoperative abdominal symptoms plus an x-ray niche, usually on the gastric side of the anastomosis. On re-exploration, no recurrent ulcer whatsoever was found, the niche being found to be merely an outpouching. This may explain a few of the recurrences at least in Goligher's series. To obviate this difficulty, we now obtain an early predischarge baseline barium study of the stomach for future reference.

4. Length of Follow-up and Per Cent of Patients Followed Up. Since our series of straight Billroth 1 resections closed on September 1, 1955, our length of follow-up study is good. However, since our patients were mainly operated upon at the King County Hospital, the percentage of follow-up study is poor. Most certainly we have not attained the 100 per cent follow-up results such as Dr. Hutchinson has. This is despite many organized efforts on our part to contact our patients. According to Dr. Volwiler, Chief of our Gastroenterology Service, we have not uncovered any new recurrences within the past year.

5. Technical Factors. We believe that technical factors, either alone or in combination, may explain why our incidence of recurrence (0.5 to 3 per cent depending on whether we include only proved cases or those with any abdominal symptoms such as persistent pain, bleeding, etc.) is lower than that reported by some others, including Dr. Hutchinson. These include:

(a) Mobilization of the duodenum by the Kocher maneuver (1903).

(b) Use of the Strauss maneuver (1952) to excise routinely the ulcer-bearing portion of the duodenum. The Strauss maneuver (*J. A. M. A.*, 149: 1095, 1952) is in my opinion a much unappreciated technical adjunct. It is most certainly one of the technical secrets of doing a Billroth 1 for duodenal ulcer. In addition, routine removal of the ulcer made possible by the Strauss maneuver, places the anastomosis closer to the neutralizing juices poured out through the ampulla of Vater.

(c) Routine ligation of the left gastric artery with high resection along the lesser curvature. The total extent of resection is not enough of a criterion if the wrong portion of the stomach is removed and a portion of the tongue of antrum lying along the lesser curvature is not removed. Furthermore, the tongue of antrum, if not removed, is vagally innervated which we have shown by dog experiments, rendering it especially ulcerogenic as far as gastrin production is concerned. In addition, if the resection does go high on the lesser curve, a major portion of the parietal cell-bearing corpus is inadvertently vagally denervated which renders our technic even less ulcerogenic.

(d) Use of non-inverting anastomosis. This technic, using a suture much like that previously described by Dr. Pierandozzi or a little like a Gambee stitch in its results, prevents anastomosis obstruction. The resultant regurgitation of alkaline duodenal juices may permit neutralization of the portion of stomach adjacent to the anastomosis where

the few stomal ulcers following Billroth I procedure in our series have recurred.

(e) Size of anastomosis. Our anastomosis is larger than that made by most other physicians performing Billroth I operations in addition to the factors listed in (d) previously. It is 3.2 cm. in outside diameter. We also always anastomose to the greater curvature portion of the stomach. Since 40 per cent of Dr. Hutchinson's cases involved anastomosis to the lesser curve by the Horsley technic (really an entirely different operation than ours and not comparable) I question not only the size of anastomosis in his patients, if it was measured, but also how many of the six recurrences were in the Billroth I anastomosis and how many in the cases of anastomosis to the lesser curvature by the Horsley technic.

(f) Extent of resection. In our opinion, this is the most usual explanation for a difference in results as far as recurrence rate is concerned. Furthermore, since dumping is, in part at least, associated with a large resection, the low incidence of dumping in Ordahl's series is circumstantial evidence for an inadequate resection. Also some surgeons "hedge" on the extent of resection when they plan to do a Billroth I because they are afraid they cannot pull the ends together. I might say parenthetically that I performed three total gastrectomies for carcinoma with esophagoduodenal anastomosis before I ever performed a Billroth I operation for ulcers. Most certainly if the esophagus can be brought to the duodenum, the stomach can.

We routinely measure the extent of resection by patterns which are later measured with the polar planimeter. Thus, a 73 per cent resection in our hands is a quantitatively measured 73 per cent resection. To my knowledge, no other group which has reported a high incidence of recurrence after Billroth I resection ever made any effort whatsoever to document quantitatively their reported extent of resection.

In conclusion, my interest in the Billroth I is, in part at least, objective since I abandoned its use by preference five years ago. This abandonment was, as I have stated, not for reasons of recurrence.

PHILIP SANDBLOM (Sweden): I worked for six years with Dr. Bohmansson in Orebro, who was one of the pioneers of the Billroth I procedure in Sweden and I continued to work with Dr. Soderlund who favored the Billroth II procedure, so I have had experience with both procedures for a long time.

In Sweden in 1932 I thought of proving the supposed advantage of the Billroth I procedure as being more physiologic, by demonstrating that the gallbladder perhaps functioned better after the Billroth I procedure. But I could just show that the gallbladder functioned exactly the same after Billroth I as after the Billroth II procedure.

To solve the question of which would be the best, several men in Sweden have collected and followed up the material from the Billroth I and Billroth II procedures and compared them. The results have shown that the Billroth I procedure gives the high recurrence rate of 7 per cent of duodenal ulcer, whereas the percentage of dumping from the Billroth II procedure has been very high.

It also was shown that there was a great difference in sex, in that the recurrence of duodenal ulcers occurred especially in men, and the dumping syndrome especially in women. Therefore, it has been recommended and is followed in many quarters to do the Billroth I procedure for duodenal ulcer in women, and the Billroth II procedure for duodenal ulcer in men. I think this is a good recommendation.

Now, of course, the introduction of vagotomy has changed the whole picture, and we have to look at it in a new manner. I think the introduction of the vagotomy which probably will reduce the recurrence rate of ulcer after the Billroth I procedure will only favor the Billroth I procedure more. I still think, however, that the Billroth II procedure should be preferred in duodenal ulcer in men.

JACK M. FARRIS (Los Angeles, Calif.): I have a collection of data on Billroth I procedures, some of which were not mentioned previously. Ordahl's series from Boston is particularly distressing, thirty-five cases followed up for less than twenty-seven months with a recurrence rate of 28.6 per cent. The addition of vagotomy, however, may reduce it to less than 1 per cent.

This makes it apparent that an operation that fails to cope with the cephalic aspects of secretory activity as well as the antral effects is bound to be met with a higher incidence of failures and should be discarded.

I was interested in reading recently about the Pima Indians, who live around Tucson. It was pointed out that ulcer is rarely, if ever, seen in these particular Indians, who live in a non-competitive communal environment. However, if you remove the Indian from this environment to the more competitive environment, he subconsciously rebels, and a duodenal ulcer may develop.

It seems to me that a consideration of these particular facts indicates that the total problem is more complex than something one might solve with a planimeter. A measurement of a certain number of square centimeters of normal stomach, no matter how ingenious, will not equate what is going on in his brain.

SIEGFRIED F. HERRMANN (Tacoma, Wash.): I have long been convinced that Dr. Harkins has the

right idea about the surgical management of duodenal ulcer. There are some things that he does differently, the most important of which, I think, is that he mobilizes the duodenum. I think it is most important to have the duodenum reach the gastric remnant, and not a gastric remnant that is mobilized to reach the duodenum. This is when unsatisfactory symptoms develop.

With Dr. Harkins' procedure a sufficiently high gastric resection can be performed to bring the duodenum to the stomach, not the stomach to the duodenum, and that is why he observes fewer recurrences. As he says, the acid-secreting portion of the stomach has been removed more effectively.

Another thing should be said about the importance of this maneuver. In a perforated ulcer perhaps it is well never to incise the peritoneum and invite infection. But here is not a perforated ulcer, but a duodenal ulcer for resection. If the duodenum is properly mobilized one can approximate the ends. But if, on the other hand, the small gastric remnant is mobilized and pulled down, then you have a straight pathway. The food that comes into the gastric remnant goes directly into the duodenum; there is immediate dumping. There is also a loss of the cardioesophageal angle so that regurgitation occurs.

I know from personal experience that in such an arrangement, if the patient lies down after eating, food runs up to his neck because of regurgitation from the loss of the cardioesophageal angle. If he stands up, it dumps down into his intestine, and he gets undesirable symptoms from that. So I think the most important thing we should remember about the difference in these results is that there probably is a difference in the employment of the Kocher maneuver in mobilizing the duodenum.

I am convinced that the Billroth I operation can be a more physiologic operation and I am also convinced that we do not improve on nature very much if we stray too far from the original pattern.

FREDERIC P. SHIDLER (Menlo Park, Calif.): My experience is not extensive, but we have been performing surgery for a few years. It seems to me that my generation is the one in which gastric resection and active surgical treatment for duodenal ulcer has come into some type of fruition.

I thought it might be of interest to notice that when I finished training and went to other hospitals, the kind of instruments that I was taught to use in an aseptic gastric resection were not available. So I had to change my technic, and this happened to be a Polya type of Billroth II operation which I did not like because it was an open anastomosis. However, I was amazed that the results were so satisfactory.

For about six years, we used this operation for duodenal ulcer and I would say with relief of patient's symptoms and adequate postoperative nutrition. We had no mortality, duodenal stump leaks, Y tubes to decompress the proximal distended loop which did not occur, and no major dumping syndromes. We then took up the Billroth I operation and for the past nine years have been using it, with satisfactory results, no perforations of suture line, no major postoperative postgastrectomy dumping syndromes, and no mortality. We removed all duodenal ulcers and have not used drainage in any case.

Then I became more interested in the physiologic aspects of gastric secretion and we are now performing vagotomies and Heineke-Mikulicz type of pyloroplasty for duodenal ulcers. Both patients and we have been satisfied with this procedure.

We have the advantage in a practical sense that we often see our patients on the street because we live in a small community, and I think that if our results were poor in any of these operations, it wouldn't be long before we would be taking different directions in our daily travel.

It seems to me that it does not make much difference what type of operation you employ, if you happen to employ it in the right manner.

I think the punch line comes in our results. In the Polya type, as I have said, we have had no recurrent ulceration that we know of. In the Billroth I operations, two patients had recurrent ulcerations within six months of each other, but there has been no other that I know of. The nutrition in the Billroth I is far superior to the Polya type. The last vagotomy-pyloroplasty we performed for bleeding duodenal ulcer after a third massive hemorrhage was followed by another hemorrhage two months later.

FREDERIC H. BENTLEY (Portland, Ore.): There is a historical side to this that should not be overlooked. In 1923 Professor Morley brought the Billroth I operation to England from Dr. Schoemaker of The Hague. Dr. Sandblom will know well Dr. Schoemaker's reputation in modifying the Billroth I procedure. From 1923 to 1939 I was with Morley as student, interne or assistant; the first paper I published in a surgical journal was in 1936 on the late results of gastrectomy in one hundred fifty or so of Morley's cases, and reviewed more than six years after the operation. That was twenty-four years ago, and I mention it only because it shows that the experience of the Manchester school is neither recent nor small.

Early in that experience, Morley found that the recurrence rate of ulcers, after a Billroth I resection for duodenal ulcer, was too high, so that he gave it up. In the period of my training I was told not to perform this operation for duodenal ulcer because recurrences were too frequent, although the results for gastric ulcer were excellent.

Several times Morley's young men, of whom I am proud to be one, would re-examine this question,

and each time would retire in confusion. Just after the war I performed two Billroth I operations for stomal ulcers. The duodenum looked so good that I thought it was perfectly safe, having performed a high resection, to anastomose back to the duodenum. In each case the result was lamentable, from recurrent ulceration. These kinds of experience make it difficult to accept Dr. Harkins' recommendation of the Billroth I operation for duodenal ulcer. It is true that the Billroth I procedure for a gastric lesion is a first class operation. It has a low mortality, and gives a pleasant result to the patient. Why is it not quite the same in Harkins' duodenal series as in Hutchinson's?

I think it is because the results of an operation for duodenal ulcer depend not only on what operation you perform as on what the patient had when you performed it. The results of an operation on a duodenal ulcer with a chronic ulcer mass, with a hole into the pancreas, are pretty good whatever you do. If, however, there is a red stippled duodenum with a lot of edema around, and a small supraduodenal diverticulum, I think you will have some poor results after any surgical procedure. For such an ulcer problem I have become more and more chary of doing anything other than a physiologic operation such as vagotomy and drainage. It is my conclusion that the difference in the results of our protagonists lies largely in the material that they are operating on, rather than in the skill with which they perform the operations, the extent of the resection, or the type of procedure.

PHILIP SANDBLOM (Sweden): I think that it is not really fair to discuss only the recurrence of duodenal ulcers which are generally evident after the Billroth I procedure, whereas the dumping syndrome you will often find only if you do a very careful follow-up study.

Dr. Wallensten's figures showing the recurrence of duodenal ulcers after Billroth I operations to be 8 per cent, I think has been reconfirmed now in Sweden, so we believe that this figure is fairly accurate.

But in the same material, Dr. Wallensten showed that the severe dumping syndrome was much more frequent after Billroth II operations, and the mild dumping syndrome occurred in 30 to 40 per cent of patients. Dr. Wallensten and others are now performing Billroth I operations on many patients who previously underwent Billroth II operations.

I am sure that with vagotomy the Billroth I operation is going to gain preference.

WILLIAM B. HUTCHINSON (closing): Dr. Kiriluk was an asset in our work because he had formerly worked with Dr. Harkins and actually performed about a third of these operations. Therefore, the technic we used was similar to the one recommended by Dr. Harkins.

We have been disturbed about this series because we like the Billroth I operation. It had many advantages, but certainly it has not worked out. We will continue to use it for other problems, such as the gastric ulcer and gastric carcinoma.

Now, as regards the age of these patients, there was only one patient under forty years of age. She was thirty-one years old and had a recurrence. The age spread and recurrence rate are just about what you would expect.

The extent of the resection was certainly adequate. We did not use a planimeter, but there are defects with using the planimeter. You cannot be absolutely sure with that either, so I do not think that the amount of resected stomach is a factor here.

In mobilizing the duodenum in each patient, the method recommended by Dr. Harkins was used. The ulcer was removed in all except one patient, and he has not had a recurrence, so that case can be eliminated.

As far as the recurrences go, of the ones that were connected by Dr. Harkins' method, two recurrences occurred. In the method of Horsely, which I believed was a little more physiologic, if you can use that term, we had four recurrences, so the ratio was just about the same for both.

I would agree that the selection of the patient has a great deal to do with the results you obtain. We selected our patients carefully, and did not operate on red, stippled duodenums. Because of our results, we are not going to perform Billroth I operations without ancillary measures for chronic duodenal ulcer.

Gastrointestinal Hemorrhage in the Aged

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DURING the past few years following the earlier studies of Mathewson [1], the Stanford University surgical service of the San Francisco Hospital has followed the empirical policy of exploring all patients over forty-five years of age in whom gastrointestinal hemorrhage is not controlled after adequate replacement of blood. While this policy has been satisfactory in general, it has been noted that in patients over sixty-five years of age, there appeared to be a higher percentage of negative explorations than in the group forty-five to sixty-five years of age.

In order to study this question, the records of all patients entering the San Francisco Hospital between July 1956 and October 1958 with a record of gastrointestinal hemorrhage were reviewed. Of the 113 patients studied during this period, fifty-three were over the age of sixty-five. Their records form the basis of this report. (Table I.)

For the purpose of this discussion, gastrointestinal hemorrhage is considered to have occurred when a patient presents a history or visible evidence of bleeding from the mouth or rectum and clinical evidence of hemorrhage confirmed by laboratory evidence of blood loss.

TABLE I
AGE DISTRIBUTION OF FIFTY-THREE PATIENTS WITH GASTROINTESTINAL HEMORRHAGE

Age	Number of Patients
66–70	11
71–75	12
76–80	12
81–85	8
86–97	10

In this group of patients, there were thirty-one males and twenty-two females. Twenty-three patients had frank hematemesis or bloody fluid was aspirated from the stomach. Thirty had grossly bloody or guiac positive stools. Twelve patients were in the hospital for other complaints when the hemorrhage occurred. Twenty-five of the patients with gastrointestinal hemorrhage died. Of this number, an autopsy was performed upon eleven. The results are shown in Table II. Twenty-eight patients survived the hemorrhage because of supportive therapy and were carefully studied by the customary methods. (Table III.) An attempt has been made to follow these patients who left the hospital up to the present time. The follow-up data are presented in Table IV. One patient who bled again has done so on three separate occasions. On each occasion he has been studied carefully without any source of bleeding being discovered.

COMMENTS

Many surgeons in this country [6] believe that uncontrolled gastrointestinal hemorrhage

TABLE II
SOURCE OF HEMORRHAGE AS DETERMINED AT AUTOPSY IN PATIENTS WHO DIED OF GASTROINTESTINAL HEMORRHAGE

Source	No. of Cases
No source of bleeding found	3
Acute hemorrhagic gastritis	1
Cardioesophageal ulcers	2
Duodenal ulcer	2
Small bowel varices	1
Carcinoma of the stomach	1
Carcinoma of the pancreas	1
Total	11

TABLE III
DISCHARGE DIAGNOSIS ON THE PATIENTS OVER THE AGE OF SIXTY-FIVE WHO RECOVERED FROM A GASTROINTESTINAL HEMORRHAGE

Diagnosis	No. of Patients
No demonstrable gastrointestinal disease	19
Diverticulosis of the colon	3
Duodenal ulcer, cirrhosis of the liver	1
Duodenal ulcer	3
Hiatus hernia	2
Total	28

TABLE IV
FOLLOW-UP DATA ON TWENTY-EIGHT PATIENTS OVER THE AGE OF SIXTY-FIVE WHO RECOVERED FROM A GASTROINTESTINAL HEMORRHAGE

Status	No. of Patients
Living, without further hemorrhage	8
Dead, other causes	9
Lost to follow-up	9
Bled again	2
Total	28

from gastric or duodenal ulceration is an indication for operative intervention. Since there is a similar tendency among surgeons treating large groups of cirrhotics, and since these two groups make up the majority of patients with massive gastrointestinal hemorrhage of unknown origin, it is apparent that many of these patients will be subjected to surgery. Such a policy in the older age group of patients will yield a high percentage of negative explorations.

It is evident from this study of fifty-three patients with gastrointestinal bleeding that the source of the hemorrhage would have been discovered in only nine had they been explored surgically. Although twenty-five of the fifty-three patients died, a mortality rate of nearly 50 per cent in this age group, it is likely that only five would have benefitted by surgical therapy.

It is extremely hazardous to set up hard and fast rules regarding the selection of surgical versus conservative management of patients with gastrointestinal bleeding. Emergency surgery as a means to control massive gastrointestinal hemorrhage is often a life-saving procedure, particularly when conservative measures have failed and when the site of bleeding can be identified and controlled at the time of surgery. Unfortunately, the site of bleeding is frequently not evident from preoperative studies or surgical exploration of the abdomen.

When the abdomen is opened and the source of the bleeding not found, the surgeon is confronted with the advisability of "blind" gastric resection. These studies would indicate that further bleeding might positively have been prevented by surgical intervention in only three of the eleven patients who bled to death and came to autopsy, and in only four of the twenty-eight who recovered without surgery. In each of these cases, the source of hemorrhage would have been apparent to an experienced surgeon. It is questionable that the three patients who died without an evident source of bleeding being found at autopsy would have benefitted by blind resection. The nineteen who recovered without demonstrable gastrointestinal disease were certainly best treated by conservative means. These observations have led us to the conclusion that blind gastric resection where no ulcer is demonstrated as advocated by Gray, Shands and Thuringer [4] is seldom indicated. In fact, our data would suggest that unless a preoperative diagnosis of the source of hemorrhage can be established in the aged patient, one would be safer to support the patient by non-operative means until the source of bleeding can be determined.

The patients who are most apt to be benefitted by emergency surgery are those with sudden massive upper gastrointestinal bleeding. In our opinion, massive bleeding must be defined and restricted to those patients whose hemorrhage is sudden and of sufficient magnitude to cause shock. Although surgical intervention may become the treatment of choice in lesser degrees of hemorrhage, this should be undertaken not as an emergency, but only after careful and exhaustive studies have demonstrated the likely site of bleeding as one which can be relieved by surgical methods.

SUMMARY

Fifty-three patients over sixty-five years of age with gastrointestinal hemorrhage have been studied. The data would suggest that gastric resection when the source of bleeding is un-

known is not as satisfactory a means of therapy as in a similar group of patients who were forty-five to sixty-five years of age.

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DISCUSSION

Ralph Loe (Seattle, Wash.): Dr. Cohn and his associates champion supportive therapy in that group of patients over sixty-five years of age whose source of hemorrhage is undetermined. This does not imply that they will not operate on an older patient who has a known duodenal ulcer not controlled by medical treatment. I am sure that they would still operate upon the latter group of patients.

The therapy for massive gastrointestinal hemorrhage appears to be reasonably well defined at the present time. I think there are relatively few surgeons who do not try conservative management for patients with massive hemorrhage. There are those, such as Stewart, who operate earlier than others, but on the whole, surgery in the presence of massive hemorrhage is generally believed to be a very serious procedure. The optimum time for surgery is when hemorrhage has ceased and the diagnosis has been established. It has been long recognized that hemorrhage in the patient over fifty years of age is much more serious than in the younger age group. This is largely due to arteriosclerosis. The loss of arterial elasticity causes a loss of compensating mechanisms in the event of serious hemorrhage. I also oppose gastric resection for hemorrhage of undetermined origin.

A closer examination for sliding hernia should be made at the time of emergency surgery for hemorrhage. I would likewise point out that the Mallory-Weiss syndrome which occurs after severe vomiting results only with a sliding hernia which produces a linear splitting of the mucosa at the entrance of the stomach into the diaphragm. There is some indication that blind gastric resection might inadvertently be beneficial in treating a sliding hernia since it eliminates pylorospasm and thus reduces gastric pressure.

Control of hemorrhage by medical means is preferable, but it would be very difficult for me to defer surgery after a sufficient period of supportive therapy with continued bleeding. We are confronted with a situation which is, of course, a matter of clinical judgement. On the other hand, I agree with Dr. Cohn that exploration in the presence of bleeding is a difficult and often frustrating thing which any experienced surgeon wishes to avoid if at all possible.

If we agree that medical management of massive hemorrhage is preferable, I would be remiss not to discuss briefly the management of massive gastrointestinal hemorrhage from the medical point of view. Costello very aptly pointed out that the mortality dropped from 25 per cent to 4 per cent in a city institution in St. Louis when adequate transfusions were given in the more aged patients. The mortality in this group (sixty-five years of age) is approximately 50 per cent. I would hasten to point out that there is no similar age group to compare mortality statistics.

I have been somewhat intrigued by the possible use of hypotensive therapy in the treatment of massive hemorrhage. Dr. Mousel, head of the Department of Anesthesia at Swedish Hospital in Seattle, believes that it might well have possibilities. The total loss of blood is reduced by hypotension. If one could partially restore blood volume and maintain a systolic blood pressure at 90 mm. Hg, it would seem possible to control bleeding to some extent. If, in addition, one used a continuous Amphogel® drip (as Woldmann has advocated) and instilled topical thrombine (advocated by Johnson), perhaps one could exert better medical control of bleeding.

Dr. Woldmann (1941) reported three deaths among 144 patients with massive hemorrhage who were treated by medical means. He used a continuous Amphogel drip and transfused patients sufficiently to keep their systolic blood pressure above 90 mm. Hg. Have we been over transfusing patients? Perhaps it might be worth our efforts to persue the medical management of these patients further. I sympathize with the surgeon who feels driven to explore the elderly patient with a massive hemorrhage of unknown origin.

Charles E. MacMahon (Seattle, Wash.): I am never quite sure of what "medical or conservative management of massive gastrointestinal bleeding" means. Since it is medical rather than surgical, it has a certain non-aggressive virtue. The use of the word "conservative" implies that more direct methods of controlling hemorrhage are radical. Perhaps the mortality rate in preoperatively undiagnosed and unlocalized hemorrhagic situations would suggest that direct control is radical.

Basically, the medical management of massive gastrointestinal hemorrhage has varied little since antiquity. When man bled he lay down to rest. If the natural reparative powers of the tissues were adequate, healing occurred; and if they were inadequate, the patient died. Replacement of blood, some methods of acid control such as the Meulengracht diet and the use of antacids have been added. Pro-Banthine® and other agents of this type are more recent additions to therapy.

Another conservative medical treatment is the use of bags to control bleeding from esophageal varices, or to attempt to control bleeding from undiagnosed cardioesophageal ulcers. The reported mortality for this conservative medical technic is 25 per cent. This mortality is directly attributable to the bag. This treatment is frequently placed in the hands of the intern or resident. I seriously doubt that we would allow the resident physician to perform a surgical procedure which produced a like mortality.

I would like to recount a case history of a type of conservative medical management of gastrointestinal hemorrhage. A man was admitted to a hospital in a semi-rural community having vomited blood. Pro-Banthine was administered as well as 500 cc. of whole blood. The family physician went out of town and did not return for five days. The patient's wife arranged to have him transferred to Seattle. Physical examination revealed an obese patient who was obviously not in shock.

The resident and I interviewed him. The following morning the patient stated that he had been fairly comfortable during the night. His buccal mucosa looked like he had been chewing "snoose," and a small amount of brown stain was apparent. When questioned the patient stated that he had not vomited, but he had "spit up a little." A Levin tube was inserted and 4,000 cc. of coffee-ground material were removed.

The resident informed me that the patient had not urinated for five days. The patient was catheterized and 1,800 cc. of urine was removed. Rectal examination revealed a massive fecal impaction which was removed.

HENRY N. HARKINS (Seattle, Wash.): The type of operation which is appropriate in each situation is important as well as the decision as to whether or not one should operate.

Herron, Jesseph and I have made a study of 600 major operations performed at King County Hospital during a period of three years. The patients in the group were over seventy years of age.

Criteria vary in these series. We used the seventy-year age minimum for inclusion in our series; Dr. Cohn used sixty-five, and some of the others even include patients as young as sixty.

The number of emergencies also fluctuates. We have a high percentage of emergencies, and a high mortality among patients treated as emergencies Our patients were in a county hospital, while some of the patients in other series were private patients.

In speaking of the proved cases of gastrointestinal hemorrhage when gastric surgery is indicated, I wish to point out that in a series of fifteen emergency gastrectomies, nine of which were for bleeding ulcer, our over-all mortality was high. Among six patients who had emergency gastrectomies for perforated ulcer there was only one death. However, five of the nine patients who had emergency gastrectomies for bleeding ulcers died, which represents a 55 per cent mortality.

These patients were seventy to eighty-four years of age and the average age was seventy-seven years. The five patients who died of bleeding ulcers were from seventy to eighty-four years of age. Two patients were eighty-four years of age. The average age was seventy-nine years.

In my opinion, whether a Billroth I, a Billroth II or a Polya is being considered, one should very carefully assess the situation before electing gastric resection as the preferred procedure in patients over seventy years of age. My conclusion is that the Weinberg procedure should be given greater use in this age group, and I wish to mention two experimental evidences for this procedure. One is the work Dr. Nyhus reported some six years ago. Dr. Lyle Hay recently reported some work with histamine in dogs which definitively compared with the Weinberg and the Dragsted procedures. When the Dragsted procedure was used (with histamine) in dogs, there was a 100 per cent incidence of stomal ulcer—with the Weinberg procedure the incidence was zero per cent. In my opinion, this certainly deserves consideration, despite the fact that both of these operations (Weinberg and Dragsted) work well clinically.

We are going to consider using gastric resection less frequently in this very old, seriously ill group of patients and using the Weinberg type procedure more often.

I wish to thank the discussers for their remarks. It is interesting that there are various attempts to control hemorrhage by the use of hypothermia and by the use of pituitrin in the experimental literature. Some clinical success with the latter has been reported by Mahoney. In addition, attempts have been made to localize the source of hemorrhage in the gastrointestinal tract by the use of tagged red cells. None of these methods have been sufficiently studied to be conclusive at present.

The rich anastomosis in the stomach wall would seem to make attempts at local control by peripheral effects unlikely. However, venous or capillary hemorrhages might be controlled by these methods.

Arteriomesenteric Duodenal Compression

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We are using the term arteriomesenteric duodenal compression to describe an intermittent obstruction of the duodenum caused by pressure of the superior mesenteric vessels on its anterior surface. This is more descriptive than the terms *duodenal ileus, arteriomesenteric ileus, duodenal stasis,* or the many other names by which this condition is known. Other causes of duodenal obstruction will not be stressed in this paper. The important features of this syndrome will be reviewed, and fourteen patients who were operated upon will be described and discussed.

ANATOMIC CONSIDERATIONS

The transverse portion of the duodenum is the most fixed part of the entire alimentary canal; it is retroperitoneal, lying between the aorta and vertebrae posteriorly and the root of the mesentery of the small bowel anteriorly. This segment of the intestinal tract is in a unique position. It is placed like a nut between the jaws of a nutcracker, with a movable anterior and an immobile posterior jaw. In quadrupeds the mesentery hangs downward and there is no pressure by the mesenteric vessels on the anterior duodenal wall. Erect posture causes a narrowing of the nutcracker's jaws and, in some instances, the mesenteric vessels compress the duodenum with sufficient force to produce intermittent obstruction. (Fig. 1.) Hence, one must visualize the normal duodenum of man as being somewhat compressed by the mesenteric vessels, and realize that the syndrome we are discussing is actually an accentuation of a normal anatomic relationship. Casts of the duodenums of cadavers made by Dwight [1] show the results of variations in mesenteric vascular pressure, from a barely perceptible indentation on the anterior wall with no obstructive element to a deep groove with almost complete obstruction of the third portion of the duodenum and marked proximal dilatation. These cases also show grooves of varying depth on the posterior wall caused by the aorta and vertebral body. Stavely [2] states that a loss of fat changes the mesentery from a broad band to a cord-like structure and increases the degree of obstruction. It is interesting to note that this obstructing mechanism may compress the left renal vein as it crosses the aorta above the duodenum in the apex of the nutcracker. Halpert [3] has described the occurrence of orthostatic albuminuria from left renal congestion in these cases. As the left spermatic and ovarian veins empty into the left renal vein, obstruction of the latter may produce a left sided varicocele in man or left pampiniform varicosities in the ovary.

Minimal pressure is sufficient to obstruct the duodenum. Dragstedt et al. [4] point out that the obstruction produced by the mesenteric vessels can usually be overcome with ease by the examiner's fingers at autopsy, a fact which has led many to question the importance of this mechanism. He wrapped the lower duodenum of dogs with wide rubber bands and found that a lethal obstruction could be produced by a pressure equivalent to a column of water 6 inches high. This minimal degree of obstruction causes no symptoms if produced in the low jejunum, ileum, or colon. In 1899 Albrecht [5] demonstrated in cadavers that a downward and backward traction on the mesentery compressed the duodenum. In 1906 Conner [6] confirmed these observations with some interesting experiments with cadavers. After removing the small intestine, he attached a cord to the mesenteric pedicle and passed it through the anus. By applying weights to the end of the cord he found that it was necessary to add 500 gm., the approximate weight

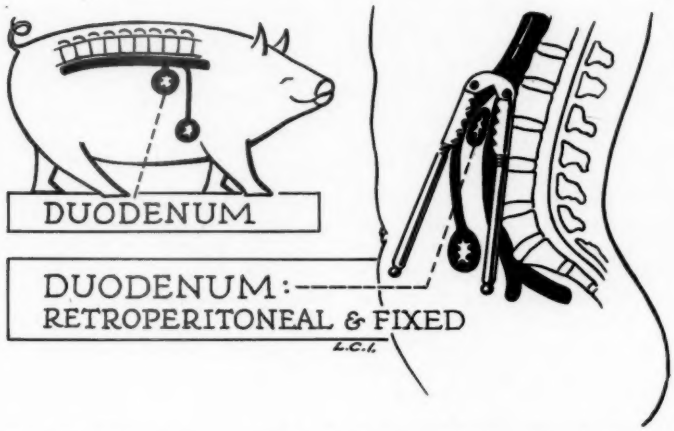

FIG. 1. Upright posture compromises the diameter of the transverse duodenum.

of the empty small intestine, to constrict the duodenum and prevent the passage of water under a pressure of 10 to 48 mm. Hg.

In the living subject, the mesenteric vessels are less mobile than the surrounding tissues and when the organs to which the arteries are distributed prolapse the vessels become the chief support of these organs and may compress the duodenum. Because of its unique anatomic position, the transverse duodenum may be obstructed with a small amount of extrinsic pressure, and it is remarkable that our upright posture has not been associated with a greater incidence of obstructive phenomena. This is due in part to the fact that the superior mesenteric vessels are directed downward and to the right, rather than lying anterior to the aorta. Also, the duodenojejunal junction may be at the right of the aorta, in which case mechanical obstruction is not possible.

Any alteration of anatomic relationships which tends to close the jaws of the nutcracker will cause some degree of duodenal compression. Visceroptosis appears to be of great importance, and is usually coupled with a loss of weight and relaxation of the abdominal wall. Lordosis may contribute. Kauffman and Gerbode [7] and others have described the onset of this syndrome following the application of a hyperextension body cast. The syndrome not infrequently makes its first appearance in chronically ill patients who have been forced to remain in a supine position for long periods. The acute form may occur following any surgery necessitating a prolonged supine position, and present a serious postoperative problem if unrecognized.

In addition to the superior mesenteric vessels, other causes which may produce external duodenal compression should be mentioned. These would include the following: imperfect rotation of the colon; a short mesentery which does not rest in the pelvis or against the anterior abdominal wall; gastric dilatation which forces the intestines downward and thus increases the mesenteric compression; a ptosed right colon which permits the middle colic artery to be directed downward so that it parallels the superior mesenteric artery causing additional pressure on the duodenum; a long transverse mesocolon with ptosis of the transverse colon; congenital or acquired bands or adhesions; a short ligament of Treitz; a narrow mesenteric attachment; or an aberrant right colic artery.

Cases of true megaduodenum have been reported in which microscopic study demonstrated a lack of myenteric plexuses in the duodenum, causing stasis in the absence of the mechanical factors described. Barnett et al. [8] point out that some of the cases diagnosed as arteriomesenteric duodenal compression are, in actuality, congenital megaduodenum.

HISTORY AND INCIDENCE

The syndrome was first described by Rokitansky [9] in 1849. Albrecht [10] described clinical cases in 1891. Duodenojejunostomy was suggested by Bloodgood [11] in 1907, and this operation was successfully performed by Stavely [2] the following year. The syndrome

was given a legitimate status by Wilkie's paper in 1921 [*12*], when he carefully described the clinical picture and outlined the treatment. In 1927 he reported sixty-four operated cases with three deaths [*13*]. In 1928 Delbet et al. [*14*] reported a procedure which joined the duodenum, pylorus and jejunum in one ostium. He and his pupils performed this operation "almost one hundred times" [*19*]. After the early 1930's, recognition of and enthusiasm for this syndrome appeared to wane. Berley and Brown [*15*] found over 100 operated cases reported in the American literature from 1920 to 1930, and only seven cases reported from 1944 to 1954. Only five cases are recorded as having received surgical therapy for this syndrome at the Los Angeles County Hospital between 1928 and 1959.

How often is this syndrome found by modern roentgenographic examination? Goin et al. [*16*] reported 1,500 consecutive upper gastrointestinal studies done in a private hospital. Five showed definite arteriomesenteric duodenal compression, and 450 showed some gross gastrointestinal lesion. He refers to a similar series of 480 cases from the Harbor General Hospital in Torrance, California, which demonstrated some gross abnormality in 173 and definite arteriomesenteric duodenal compression in two.

The many names given to this syndrome have undoubtedly added confusion to the various hospital records. However, while the exact incidence is difficult to establish, there must be a happy medium between the many cases diagnosed and operated upon in the 1920's and the dirth of reported cases in subsequent decades. We believe that the condition, while uncommon, exists as a clinical entity and will discuss some of the criteria for diagnosis and treatment, and analyze fourteen operated cases.

DIAGNOSIS

Symptoms. The classic picture of chronic arteriomesenteric duodenal compression is represented by an individual with a long-standing history of a postprandial epigastric fullness which is followed by epigastric pain. As the pylorus becomes incompetent the food regurgitates from the obstructed duodenum into the stomach, nausea develops, the patient vomits bile-stained material, and the discomfort is relieved. As this symptom complex progresses, the patient becomes afraid to eat heartily, loses more weight, and a vicious cycle is instituted. Frequently this picture is not as clear-cut as described. Vague and ill-defined epigastric discomfort after eating may be the only complaint. This may be associated with anorexia, malaise, nervousness, easy fatigability, migraine, marked mental depression and emotional instability. Reichert [*17*] has described patients in whom this condition was not recognized who were sent to him because of their neurological symptoms. Appropriate treatment to relieve the duodenal obstruction resulted in a disappearance of the neurological symptoms.

The acute form presents the picture of high intestinal obstruction with gastric dilatation and vomiting of bile-stained material. The latter feature distinguishes it from pyloric obstruction.

It is important to mention that peptic ulcer may be present in approximately 25 per cent of patients with this condition, and may confuse the clinical picture. The gallbladder is frequently incriminated as the cause of the symptoms. On occasion, abdominal exploration has been performed to determine the cause of the patient's symptoms and the condition has not been recognized and treated. This occurred five times in our series. These explorations were performed by surgeons other than the authors.

Physical Examination. These patients are usually slender, asthenic and visceroptotic. Eighty per cent of patients in Bockus' series were underweight [*18*]. Except in cases with acute obstruction when a succussion sound may be heard, little is found on routine examination. Relief of symptoms obtained by a knee-chest or prone position suggests the diagnosis.

Roentgenographic Examination. A positive roentgenographic diagnosis is indicated by: (1) dilatation of the proximal duodenum down to the midline, or a delay in the passage of barium across the midline (Fig. 2); (2) churning of barium in the duodenum as seen by fluoroscopy, which in itself it not always diagnostic; (3) a disappearance of the duodenal retention when the patient is placed in a prone position or when manual pressure is exerted on the lower abdomen in a cephalad direction while the patient is in a prone position (Hayes' maneuver); (4) a flat film can be diagnostic in

FIG. 2. Duodenal distention proximal to the "cut-off."

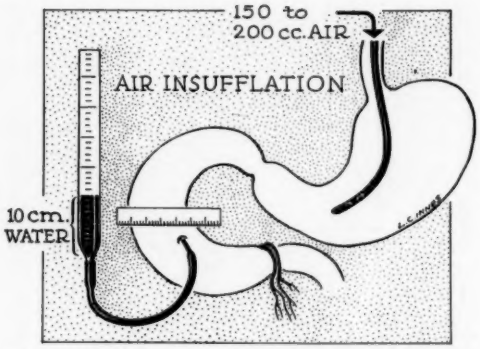

FIG. 3. Diagram of air insufflation technic used at the operating table.

the acute case, and may demonstrate gross gastric and duodenal distention with minimal air in the distal bowel.

It must be stressed that this condition is intermittent in nature, and for this reason the absence of typical roentgenographic signs at any one examination does not eliminate the existence of the syndrome. A negative result may also be obtained if the referring doctor fails to mention the possible presence of the condition, and if the roentgenologist is unfamiliar with the methods used for its demonstration. In our experience, the condition is best shown at fluoroscopy with the patient leaning backwards at a 30 degree angle from the upright position. The radiologist allows the duodenum to fill with barium and then pushes the stomach upward to eliminate its shadow which otherwise obstructs the view of the third portion of the duodenum. Oblique views are sometimes needed. These simple additions to the usual routine of an upper gastrointestinal study may be very rewarding. It is important to remember that some roentgenologists are prone to make this diagnosis more frequently than others, and a mild degree of obstruction may be seen on occasion in normal people. The surgeon must be certain that these minimal findings do not encourage him to use unnecessary radical treatment or to overlook other more significant pathology.

Diagnosis at the Operating Table. Air insufflation: The normal duodenum is capable of minimal distention because of its investment in a strong peritoneal covering and its muscle tone. With intermittent obstruction, the peritoneum is gradually stretched and the muscle tone reduced until the organ reaches a large diameter, especially when subjected to internal pressure [4]. This increased diameter may not be readily apparent at surgery unless the duodenum is filled with air or fluid, and the condition may be overlooked by the casual observer. In an effort to distinguish the normal from the intermittently obstructed duodenums, we have instilled air into the stomach by a Levin tube when the abdomen is open.

1. Studies on normal patients: Simple instillation of air was performed in fifty people with no duodenal obstruction; 150 to 300 cc. of air was instilled through the intragastric tube by the anesthesiologist. It was noted that the duodenum would distend only slightly, and that the air would pass slowly from the duodenum to the jejunum, taking from two to six minutes to empty. Following these initial observations, more precise data were obtained in ten additional patients without mechanical obstruction. A needle was introduced into the kocherized duodenum and connected to a water manometer. Air was then introduced through the Levin tube until the intraduodenal pressure

FIG. 4. Air insufflation study of a normal duodenum.

measured at least 10 cm. of saline. The diameter of the duodenum was measured before and after air insufflation. (Figs. 3 and 4.) The results are given in Table I.

2. Studies on patients with arteriomesenteric duodenal compression: Air insufflation has been performed in seven of fourteen patients in our series. Intraluminal pressures were not taken. In all of these cases the duodenums have been found to distend markedly down to the level of the superior mesenteric vessels, reaching a much greater diameter than occurs in the normal, and to remain distended until the root of the small bowel is elevated anteriorly. This maneuver was followed by a rapid escape of air into the jejunum which was sometimes forcible enough to be audible. The results are given in Table II.

The most important finding in these air insufflation studies is the marked distention occurring in patients with true mechanical obstruction as contrasted with the minimal change in diameter in the normal individuals. There was an average increase in diameter of only .95 centimeters in the normal, as compared with 4.8 centimeters in the patients with arteriomesenteric duodenal compression. In analyzing the results of this preliminary study, a compatible clinical picture or the surgical finding of a duodenum of 5 cm. or more in diameter should make the surgeon suspicious of the presence of this syndrome. Air insufflation studies demonstrating an increase in diameter of over 3 or 4 cm. would indicate an intermittent duodenal obstruction.

TABLE I

RESULTS OF DUODENAL INSUFFLATION IN TEN NORMAL PATIENTS*

Case No.	Amount of Air (cc.)	Intraduodenal Pressure (cm. of saline)	Pre-insufflation Diameter (cm.)	Post-insufflation Diameter (cm.)
1974 949	200	10	3.5	4.0
1957 637	250	11	4.0	5.5
1065 142	200	11	3.5	4.5
1824 471	300	13	3.0	3.5
1678 075	350	10	4.0	5.0
1976 997	300	10	4.0	5.5
1466 273	150	20	3.5	4.5
1409 344	150	20	4.0	5.5
1873 660	200	15	3.5	4.0
1648 391	200	12	4.5	5.0
Averages...	230	13.2	3.75	4.70

* The average increase in diameter was 0.95 cm., the greatest increase was 1.5 cm., and the smallest increase was 0.5 cm.

TABLE II

RESULTS OF DUODENAL INSUFFLATION IN SEVEN PATIENTS WITH ARTERIOMESENTERIC DUODENAL COMPRESSION*

Case No.	Amount of Air (cc.)	Preinsufflation Diameter (cm.)	Postinsufflation Diameter (cm.)
C 93947	200	6.0	10.0
C 93142	225	3.5	8.5
C 66723	180	4.5	9.5
133494	200	5.0	11.0
58-10372	300	10.0	15.0
D 63903	225	8.0	12.0
372251P	200	10.0	15.0
Averages...	218	6.7	11.5

* The average increase in diameter was 4.8 cm., the greatest increase was 6.0 cm., and the smallest increase was 4.0 cm.

The retention of air for a longer period in the cases of obstruction is also of significance. In three of our patients the diagnosis would have been missed if air insufflation had not been performed. In addition to this there were five abdominal operations in four patients in this series in which a diagnosis was not established, and could have been made by the use of this procedure. We recently discovered that Wilkie [*12*] used simple air insufflation in 1921, but abandoned it later, stating in 1927 [*13*] that is was not trustworthy. We regard the procedure as outlined as an adjunct to diagnosis which has proved useful in our experience.

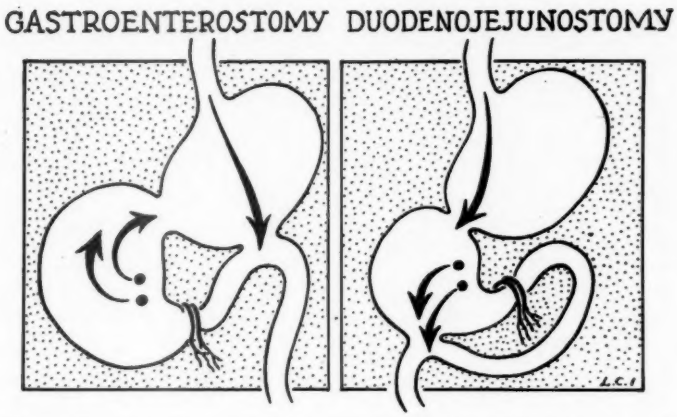

FIG. 5. Gastroenterostomy necessitates a regurgitation of bile and pancreatic juice into the stomach, which is eliminated by a duodenojejunostomy.

Bulge of duodenum through the mesocolon: Kellogg et al. [19] point out the significance of observing a duodenum which bulges downwards through the transverse mesocolon. This may be present even though the duodenum does not appear to be distended when viewed from the supracolic position.

Adhesions: Congenital or postinflammatory bands or adhesions should be ruled out by inspection and palpation.

Aberrant vessels: These must be searched for as a cause of duodenal compression.

Internal palpation: If surgery for peptic ulcer is being performed, an obstructing mechanism may be demonstrated by passing a finger within the duodenum down to the level of the vessels at the time of pyloroplasty or before the duodenal stump is closed. Some experience is necessary in evaluating the degree of pressure which is abnormal, and one should not expect to find a marked occlusion even in a well established case of arteriomesenteric duodenal compression.

Biopsy: Biopsy of the duodenum to determine the presence or absence of myenteric plexuses would be of value in differentiating aganglionic megaduodenum from arteriomesenteric duodenal compression.

TREATMENT

Medical. Any discussion of treatment should emphatically state that arteriomesenteric duodenal compression is a medical disease until proved otherwise. Pool [20] has pointed out that medical treatment is usually adequate in patients with a short history and only a moderate degree of duodenal stasis as demonstrated by roentgenograms. In acute cases a prone position with the foot of the bed elevated or a knee-chest position may open the nutcracker and afford relief of the obstruction. If this fails one must aspirate the stomach and consider the use of a long feeding tube. Electrolyte replacement is given as indicated.

In chronic cases, positional therapy as previously described is indicated. Visceroptosis is treated by physiotherapy, abdominal exercises and abdominal support. Feedings should be frequent and of a bland, low-residue, high-caloric type. If a gain in weight occurs, medical treatment is often successful. Constant reassurance and even psychotherapy may be needed. During the seven years and nine months of this study, one of us observed seven cases treated as outlined previously to the point where they were entirely asymptomatic.

Surgical. Surgical therapy is indicated when a trial of medical treatment has failed. In general, surgery has been found necessary in those patients with: (1) history of uncontrolled indigestion for many years; (2) progressive loss of weight and strength while on medical treatment; (3) pronounced dilatation and stasis in the duodenum as seen on roentgenograms.

If surgery is indicated for uncomplicated arteriomesenteric duodenal compression, we believe that a retrocolic duodenojejunostomy is the method of choice. Figure 5 will illustrate

TABLE III

Case No.	Sex, Age (yr.)	Weight Loss (lb.)	Duration of Illness (yr.)	Symptoms	Roentgenograms	Previous Surgery (other surgeons)	Our Findings	Air Insufflation (cm.)	Our Procedure	Result
C 93947	M., 34	15	17	Postprandial vomiting with bile; nervousness	Arteriomesenteric duodenal compression; duodenal ulcer	None	Arteriomesenteric duodenal compression and small acute duodenal ulcer	6 to 10	Vagotomy and pyloroplasty; duodenojejunostomy	Excellent
C 93142	M., 40	12	9 mo.	Postprandial vomiting with bile; nervousness	Negative upper gastrointestinal series	Exploration by osteopath indicating a diaphragmatic hernia; no surgical treatment for this; arteriomesenteric duodenal compression suspected from history given us	No hernia present; tract normal until air insufflation performed and gross distention demonstrated down to the vessels	3.5 to 8.5	Duodenojejunostomy	Excellent
C 66723	F., 48	45	40	Postprandial vomiting with bile; nervousness	Arteriomesenteric duodenal compression	Gastrojejunostomy at age 22 yr., no improvement; vagotomy at age 43 yr. with marked increase in signs and symptoms; at this time gastrojejunostomy was taken down	Marked arteriomesenteric duodenal compression	4.5 to 9.5	Duodenojejunostomy; pyloroplasty because of previous vagotomy	Excellent
372251P	M., 43	20	12 to 15	Postprandial vomiting with bile	Arteriomesenteric duodenal compression	Exploration in 1955; no diagnosis or treatment. Exploration in 1957, diagnosis was arteriomesenteric duodenal compression and chronic gallbladder disease with stones; treatment was gastrojejunostomy, cholecystectomy and vagotomy	Megaduodenum down to the vessels "like a second stomach"	10 to 15	Gastrojejunostomy; duodenojejunostomy; pyloroplasty because of previous vagotomy	Excellent
1808979	M., 64	15	6 mo.	Vague epigastric distress for 6 mo., and two days of severe epigastric pain with vomiting of bile; diagnosed as acute gastric dilatation	Read as gastric dilatation from pyloric obstruction	None	Dilated stomach; chronic duodenal scarring without pyloric obstruction; duodenum markedly dilated down to mesenteric vessels	Not performed	Sleeve resection; pyloroplasty; duodenojejunostomy	Improved; some epigastric distress; chronic alcoholic, and difficult to evaluate
133494	F., 49	20	18	Attacks of postprandial vomiting, bile stained; attacks of epigastric pain relieved by ulcer treatment which did not stop the vomiting; marked nervousness	Chronic duodenal ulcer; no other abnormality	Hysterectomy; appendectomy	Small chronic duodenal ulcer, no pyloric obstruction; duodenum not remarkable until kocherized and insufflated which showed gross distention down to the vessels; incidental jejunal diverticulum	5 to 11	Vagotomy; pyloroplasty; duodenojejunostomy; resection of jejunal diverticulum	Excellent
5810372	F., 59	20	8	Gaseous distention after eating any foods; rare vomiting; severe nervousness	Normal upper gastrointestinal series; cholangiogram (after cholecystectomy) showed slight common duct dilatation	Gallbladder removed in 1951 without relief of signs and symptoms; common duct not explored	Lower half centimeter of common duct narrowed to 2 mm., only slight distention of common duct; duodenum appeared only slightly dilated on initial examination; when adhesions were freed it measured 10 cm.	10 to 15	Sphincteroplasty; duodenojejunostomy	Excellent
897 090	F., 58	30	3	Back and epigastric pain partially relieved by vomiting and prone position for 3 yr.; 24-hr. history of hematemesis and melena	Gastric ulcers on lesser curvature; "atonic duodenum"	None	Three active gastric ulcers, one with a clot in a central vessel; marked duodenal distention proximal to the vessels; incidental adenoma of lower pole of left adrenal	Not performed	Billroth subtotal gastrectomy; duodenojejunostomy; resection of adrenal adenoma	Excellent

TABLE III (*Continued*)

Case No.	Sex, Age (yr.)	Weight Loss (lb.)	Duration of Illness (yr.)	Symptoms	Roentgenograms	Previous Surgery (other surgeons)	Our Findings	Air Insufflation (cm.)	Our Procedure	Result
960 239	F., 56	33	Chronic, 12 Acute, 6 mo.	Repeated vomiting after meals bile-stained for 12 yr.; more marked last 6 mo.	"Large flabby duodenal bulb"	None	Duodenum dilated to 7 cm. down to vessels without air; cirrhosis of the liver	Not performed	Anterior gastrojejunostomy	Relief of obstructive symptoms; died 7 mo. postoperatively of hepatic failure and a stroke
1444 061	F., 61	"Moderately cachectic"	15	Recurrent postprandial bile-stained vomiting	Arteriomesenteric duodenal compression; duodenal ulcer	Oophorectomy in 1924; perforated duodenal ulcer closed in 1953	Chronic duodenal ulcer; marked arteriomesenteric duodenal compression, 10 cm. without air insufflation; duodenum four times normal size down to vessels	Not performed	Vagotomy and posterior gastrojejunostomy	Excellent
1736195	M., 55	"Thin"	15	Copious postprandial bile-stained vomiting and epigastric distress; two bleeding episodes 1942 and 1948, melena; acute pancreatitis in 1957; severe psychoneurosis	Arteriomesenteric duodenal compression	None	Old duodenal scarring, chronic ulcer; duodenum 10 cm. in diameter down to the vessels without air insufflation	Not performed	Vagotomy; pyloroplasty; duodenojejunostomy	Much improved; vague distress "only when nervous"; severe psychoneurotic
D 63903	F., 34	20	2	Right upper quadrant and epigastric pain and nausea after eating; occasional vomiting, bile-stained	Upper gastrointestinal series negative nonfunctioning gallbladder	Appendectomy and hysterectomy	Duodenum larger than normal before insufflation and markedly enlarged down to the vessels after insufflation; gallbladder showed mild chronic inflammation	5.5 to 10	Duodenojejunostomy; cholecystectomy	Excellent
1014-50	F., 51	30	15	"Tension in stomach"; cramping upper abdominal pain not definitely related to meals	"Pyloric obstruction, gross gastric dilatation" in 1950; "dilated duodenum, backwash of barium" in 1952; "gastric polyp" in 1959	Cholecystectomy, no relief of symptoms	Hypertrophy of stomach and duodenum in 1950, latter 8 cm. in diameter without air; after adhesions freed gross dilatation down to vessels; jejunum collapsed; carcinomatous polyp and dilated duodenum as before in 1959	Not performed	Posterior gastrojejunostomy in 1950; gastrojejunostomy taken down, wedge resection of gastric polyp; duodenojejunostomy in 1959	No relief from gastrojejunostomy; excellent following duodenojejunostomy
R 43590	F., 31	Overweight 10	17	Fullness in upper abdomen "like a tight girdle"; nausea following meals with occasional vomiting	Arteriomesenteric duodenal compression	Appendectomy and oophorectomy	Marked dilatation of duodenum proximal to vascular obstruction of fourth portion	Not performed	Duodenojejunostomy	Excellent

why better duodenal drainage is obtained by this procedure than by gastroenterostomy. We see no indication for gastric resection for this condition, and, as will be mentioned later, there may be a real hazard associated with this operation. We have had no experience with the method of mobilizing the distal duodenum and allowing it to be displaced downward as described by Strong [21]. It may prove to be technically more simple and as effective as duodenojejunostomy.

CASE REPORTS

This series is composed of fourteen patients with arteriomesenteric duodenal compression who have been treated surgically over the past seven years. Nine of these are private patients, four were operated upon at the Los Angeles County Hospital, and one at the White Memorial Hospital, Los Angeles. A detailed description of these cases is given in Table III and the salient features summarized in Tables IV and V.

TABLE IV

A. Age		
Oldest	64	yr.
Youngest	31	yr.
Average	49	yr.
B. Sex		
Male	5	
Female	9	
C. Weight		
Preoperatively eleven of the fourteen patients were 12 to 45 pounds below normal, averaging 23.6 pounds. The two not weighed were stated to be "thin" in one case and "moderately cachectic" in the other. One patient was 10 pounds overweight.		
D. Duration of symptoms		
Longest	40	yr.
Shortest	6	mo.
Average	11.9	yr.
E. Symptoms		
Postprandial vomiting (bile and food)	13	
Moderate to severe	9	
Occasional	4	
Symptoms of peptic ulcers (all patients had concomitant peptic ulceration at surgery)	5	
Moderate to severe nervousness	5	
Vague upper abdominal distress	2	
Signs and symptoms of biliary tract disease in addition to symptoms of arteriomesenteric duodenal compression (one patient had stricture of lower end of common duct and another chronic cholecystitis)	2	

TABLE IV (*Continued*)

F. X-ray diagnosis	
Recognized preoperatively by x-ray examination	7
Not recognized preoperatively by x-ray examination	7
X-ray reports in cases in which arteriomesenteric duodenal compression was overlooked	
Normal upper gastrointestinal study (one preoperative diagnosis of common duct stricture, present at surgery) (one patient had previous diagnosis of diaphragmatic hernia and clinical picture of mesenteric ileus; neither was demonstrated by preoperative x-ray)	2
Pyloric obstruction	2
Duodenal ulcer (no mention of arteriomesenteric duodenal compression in gastrointestinal study; duodenal ulcer present at surgery)	1
Gastric ulcer, atonic duodenum (gastric ulcer present at surgery)	1
"Flabby duodenal bulb"	1
G. Air insufflation	
Performed	7
Successful	7
Positive gastrointestinal roentgenograms, diagnosis confirmed by air insufflation	3
Negative gastrointestinal roentgenograms, diagnosis would have been missed without air insufflation (one patient had duodenal ulcer, one patient had stricture of lower end of common duct, in addition to arteriomesenteric duodenal compression)	3
Negative gastrointestinal roentgenograms, non-functioning gallbladder. At surgery duodenum found to be slightly enlarged before insufflation, and enlarged two to three times normal size after insufflation, confirming diagnosis	1
H. Previous operations for upper abdominal symptoms	
Five operations on four patients. Diagnoses were:	
Duodenal ulcer (one patient) (arteriomesenteric duodenal compression overlooked at two previous operations)	2
Diagnosis missed, exploration only	2
Cholecystectomy without relief of symptoms (arteriomesenteric duodenal compression overlooked)	1
I. Results of five gastroenterostomies for arteriomesenteric duodenal compression	
Results good	2
Results poor	3
Reoperated and duodenojejunostomy performed with good results	3

TABLE IV (*Continued*)

J. Results of twelve duodenojejunostomies	
Excellent results	11
Questionable results (patient definitely improved, but chronic alcoholism makes evaluation difficult)	1
K. Peptic ulcer disease occurring with arteriomesenteric duodenal compression in a total of five patients	
Duodenal ulcers	
Chronic	3
Acute	1
Gastric ulcers.	
Acute	1
L. Increase in symptoms after vagotomy occurring in three patients	
Made worse by vagotomy and relieved by duodenojejunostomy	2
Symptoms improved by gastroenterostomy and vagotomy	1
M. Mortality	0
N. Over-all follow-up study:	

Case No.	Gastrointestinal Complaints	Weight Regained	Normal Activity Resumed	Duration of Follow-Up Study
C 93947	None	Yes	Yes	5 yr., 1 mo.
C 93142	None	Yes	Yes	2 yr., 6 mo.
C 66723	None	Yes	Yes	7 yr., 9 mo.
37-22-51P	None	Yes	Yes	1 yr., 3 mo.
133494	None	Yes	Yes	8 mo.
5810372	None	Yes	Yes	1 yr., 8 mo.
897090	None	Yes	Yes	8 mo.
1444061	None	Yes	Yes	9 mo.
D 63903	None	Yes	Yes	6 mo.
101450	None	Yes	Yes	7 mo.
R-43590	None	Yes	Yes	1 yr., 2 mo.
960 239	None	Ascites	Died of stroke and cirrhosis	7 mo.
1736 195	Vague distress "only when nervous" much improved; severe psychoneurotic	Yes	Yes	2 yr., 8 mo.
1808 979	Some epigastric distress, less severe; chronic alcoholic	Yes	Yes	10 mo.

TABLE V
SUMMARY OF FOLLOW-UP STUDY

Average duration of follow-up study	1 yr., 11 mo.
Shortest follow-up study	6 mo.
Longest follow-up study	7 yr., 9 mo.
Weight regained	14
Activity resumed	13
Died of other causes	1
No abdominal complaints	12
Occasional distress, marked improvement	1
Mild improvement	1

COMMENTS AND CONCLUSIONS

The following conclusions have been drawn from our experience with the fourteen cases in this series and a review of the literature. These conclusions are based on a small series of cases, and will be disproved or upheld only after further studies are made.

Early enthusiasm in the diagnosis and surgical management of this syndrome has resulted in skepticism as to the existence of this form of duodenal obstruction. We believe that arteriomesenteric duodenal compression, while rare, does exist as a clinical entity which requires treatment. The diagnosis is frequently missed because of the vagueness of the history and lack of findings on routine physical and roentgenographic examinations. A high index of suspicion is advocated in patients with vague upper abdominal complaints.

The radiologist should be alerted to the possibility of this syndrome in order to obtain the most satisfactory upper gastrointestinal study. Repeated examinations may be necessary to reach a diagnosis. Even with multiple studies the condition may be unrecognized by roentgenograms and demonstrable at surgery. Not all roentgenograms showing mild obstruction are indicative of a condition causing symptoms or requiring treatment.

Air insufflation studies and internal and external palpation of the duodenum should be performed at the operating table when any doubt exists as to the presence of this condition. In five instances in this series previous abdominal operations by other surgeons failed to demonstrate the existing arteriomesenteric duodenal compression, and the symptoms were unrelieved until duodenojejunostomy was performed.

We believe that this condition should be excluded before a choice of operative procedures is made in every case in which surgery for peptic ulcer is being considered. This is especially important when one considers the frequency with which the two conditions coexist. Given a patient with a surgical peptic ulcer and an unrecognized arteriomesenteric duodenal compression, it is both clinically and theoretically true that vagotomy and pyloroplasty alone will aggravate the symptoms due to arteriomesenteric duodenal compression. The resulting decrease in gastric and duodenal motility pro-

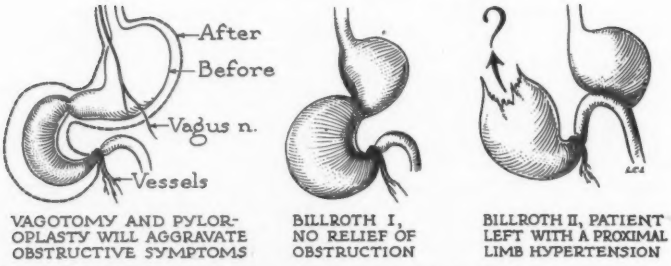

FIG. 6. In coexisting peptic ulcer and arteriomesenteric duodenal compression, the operations illustrated are unsatisfactory for the reasons shown.

FIG. 7. A preoperative film (not our patient) showing a normal duodenal loop and duodenal ulcer.

FIG. 8. A postoperative film on the same patient soon after vagotomy and gastroenterostomy showing the appearance of an arteriomesenteric duodenal compression after vagotomy.

duced by vagotomy will accentuate the duodenal obstruction. (Fig. 6.) There are reports of the syndrome making its first appearance following vagotomy. (Figs. 7 and 8.) If a Billroth II resection is chosen, the proximal limb hypertension left untreated is not desirable and could conceivably result in or contribute to a perforation of the duodenal stump. (Fig. 6.) A Billroth I operation would, of course, fail to relieve the duodenal obstruction. (Fig. 6.)

When the surgeon realizes that this combination of problems coexist, the surgical procedure is distinctly modified. In any event, duodenojejunostomy, or duodenal mobilization and downward displacement [*21*] must be performed. The ulcer may then be treated

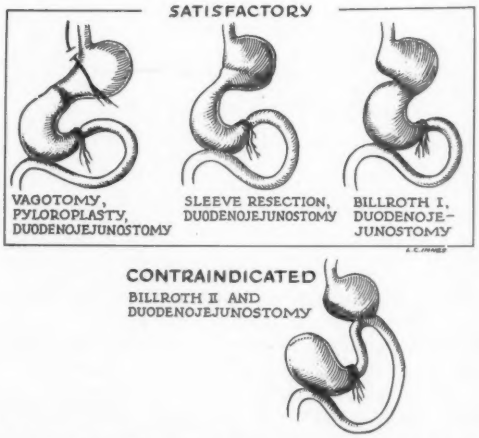

FIG. 9. Surgical therapy in coexisting peptic ulcer and arteriomesenteric duodenal compression.

by either vagotomy and pyloroplasty, sleeve resection, or a Billroth I resection. A Billroth II added to duodenojejunostomy would be clearly contraindicated as it would set the stage for a gastrojejunal ulcer. (Fig. 9.)

Failure to recognize this associated condition may account for the persistence of signs and symptoms in some patients following surgery for peptic ulcers. It is suggested that such patients be re-evaluated with arteriomesenteric duodenal compression in mind as a possible cause for their poor response to surgery for ulcers.

SUMMARY

1. Fourteen surgically treated patients with arteriomesenteric duodenal compression are presented. Twelve have been relieved of digestive symptoms, and two are improved.

2. A high index of suspicion is advocated in evaluating patients with vague abdominal complaints of long-standing.

3. Methods of diagnosing this condition at the operating table are discussed. In four cases, five abdominal operations by other surgeons had failed to demonstrate the condition. Air insufflation has been of value in our hands in recognizing this syndrome.

4. Diagnosis by roentgenogram is not certain. The condition may be overlooked or diagnosed too frequently.

5. This syndrome should be considered when surgery for peptic ulcers is indicated. The presence of arteriomesenteric duodenal compression will alter the surgical procedure used in the treatment of patients with ulcers. Patients who have had a poor response to surgery for ulcers should be re-evaluated with this condition in mind.

6. This clinical entity should be treated surgically if medical treatment fails. Conservatism in both diagnosis and treatment must be stressed.

7. The accuracy of the conclusions drawn in this preliminary paper must be determined by further studies.

Acknowledgment: We wish to express our appreciation to Dr. Ralph Thompson, Senior Surgical Resident at the Los Angeles County Hospital, for his assistance in the air insufflation studies.

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DISCUSSION

Gordon K. Smith (Los Angeles, Calif.): Arterial mesenteric duodenal compression is a definite clinical entity. During the early part of the twentieth century, numerous reports regarding this symptom complex were made and various

surgical procedures suggested. The pendulum then swung to the other extreme and little attention has been paid by the surgical profession to this disease since 1930. It has been our personal experience that the majority of these patients are asthenic and visceroptotic. Many of them also have a large functional overlay. Typical roentgenographic findings are not invariably associated with the subjective signs. Therefore, it is possible, as Dr. Jones points out, that our index of suspicion regarding this condition is extremely low. The anatomic studies that Dr. Jones and his group have made are unique. His figures showing the marked difference in diameter between the so-called normal and pathologic duodenum, as well as the increase in size with constant air insufflation, are interesting. I should like to ask Dr. Jones whether or not patients in whom these measurements were made were studied as to the type of preanesthetic medication, anesthesia used, and drugs administered during the procedure by the anesthetist. It is a well known physiologic fact that the tonus of the bowel is influenced either by stimulation or inhibition of the parasympathetic or sympathetic mechanisms, as in spinal anesthesia. I am sure that continued studies are planned in further pursuit of these preliminary observations.

Dr. Jones states that only 50 per cent of the patients in his group had sufficient roentgenographic evidence preoperatively to confirm the diagnosis. A recent report by Kaiser and Shumaker shows that practically all of eighteen patients treated surgically had sufficient obstruction to allow the condition to be diagnosed radiographically. It also would seem that chronic duodenal obstruction might contribute to stimulation of the antrum, which in turn might lead to gastric or duodenal ulcer formation. In Dr. Jones' series, there were four patients with duodenal ulcer and one with gastric ulcer, or a combined instance of some 35 per cent. Interestingly enough, there were no instances of duodenal or gastric ulcer in Dr. Kaiser's series. In our experience, we have not recognized instances of this phenomenon occurring in association with duodenal or gastric ulcer.

Almost 40 per cent of the cases reported by Kaiser developed in patients with no previous history of gastrointestinal complaints and presumably upon the basis of confinement to bed because of illness, injury, or surgical procedure. In Dr. Jones' series this, apparently, was not as noticeable, as the shortest duration of symptoms in any of his patients was six months. Therefore, it would appear that there is a great difference of opinion among those who are well aware of the incidence of this disease as to the diagnostic criteria necessary for surgical intervention. There is unanimity of opinion, however, that the ideal surgical procedure is duodenojejunostomy. It also seems reasonable to assume that some patients with this disease have been successfully treated with a Billroth II type of procedure. If one considers the size of the normal common bile duct and pancreatic duct, it would seem that the closed loop syndrome, described by Dr. Jones, would be rare in occurrence after a Billroth II operation. High-grade obstruction would have to develop in the transverse portion of the duodenum to produce a caliber less than the normal diameters of the ducts.

I believe that Dr. Jones' anatomic studies and the excellent analyses of his results should increase an awareness of this important condition. There is no question that the performance of vagus section and pyloroplasty or a Billroth I procedure would aggravate a mild pre-existing condition.

MILLARD S. ROSENBLATT (Portland, Ore.): In 1944 I published a paper which considered various phases of duodenal obstruction. I included three cases in this category in that paper. Two of these cases were treated by a sort of reverse Kocher procedure, inasmuch as we freed the duodenojejunal flexure and mobilized it. They were cured by this. The third required a duodenojejunostomy.

I have not seen a single case which demanded surgery since 1944, so I cannot be included in the group that regards this as a common syndrome, or one that is optimistic about performing this for most cases. Most of the patients I have seen since then with a similar syndrome have been improved by weight gain, positioning and other medical treatment. If this situation does arise, the possibility of an acute duodenojejunal angle should be considered. The freeing of that angle and the lowering of the gradient, may be a factor in its repair.

WERNER E. ZELLER (Portland, Ore.): My interest in this subject goes back to 1954. The wife of a physician in one of the communities near Portland had been bothered for many years with pain in the right upper quadrant, nausea, vomiting and distress. She was thirty-four years of age and the mother of four children. Pain radiated around into her back, and she had had gallstones since the age of twelve years. When she was twenty-four years of age, or twelve years before I saw her, she underwent cholecystectomy. This did absolutely nothing for her symptoms, and for twelve years her husband believed that she was psychoneurotic.

At that time we were hearing a lot about stenosis and fibrosis of the sphincter of Oddi, and I thought this patient had fibrosis of the sphincter of Oddi. Upper and lower gastrointestinal studies were negative except for a slight dilatation of the duodenum, which was not considered significant at the time.

The patient was taken to surgery, and in the course of exploration I opened the common duct and explored it. I was able to introduce a probe,

and the smallest dilator went down through the sphincter with a little difficulty, but it did not go down tightly enough to completely satisfy me. On further examination I found a situation which Dr. Jones has described. A short, tight ligament of Treitz was binding the fourth portion of the duodenum in such a fixed position that it was definitely compressed.

I freed this entire area, cutting the ligament of Treitz as Dr. Rosenblatt had described, allowing the duodenum to move caudad, and by mobilizing this area from both the infra and supra transverse mesocolon area, was able to get three fingers underneath and free this area. The patient's symptoms were completely relieved after surgery.

About eight months later the wife of this same doctor's partner came in to see me. She was thirty-four years of age, the mother of three children and four months pregnant at the time. She had had a history similar to that of the other patient. She underwent cholecystectomy in 1953 following the birth of her third child. Every time she became pregnant her symptoms were aggravated. This, I think, was attributable to the fact that her appetite became voracious at that time, and she would eat huge meals. I believe that this would fill the duodenum rapidly and increase the angulation of the duodenojejunal junction.

At any rate, the gallbladder was removed without relief of symptoms. Over a year later she underwent exploration of the common duct and cutting of the sphincter of Oddi, but had no relief of symptoms. In addition to pain in the right upper quadrant, nausea, vomiting and pain radiating to the back, she had another pain which went up toward the left shoulder.

The initial diagnosis was pancreatitis. I suspected a possibility of a common duct stone which was not found during her previous operation, and I vaguely considered the syndrome present in the other patient, but not strongly.

Surgery demonstrated that she had a problem almost identical to that of the first patient, and on freeing this duodenojejunal junction and mobilizing the fourth portion of the duodenum her symptoms, including pain in the left shoulder, ceased.

This patient's symptoms were so severe that she was taking Demerol® to the extent that she was practically addicted. Most of the narcotics that her husband had prescribed in the preceding year were given to her because of her severe distress. Her baby was delivered at term, she has had one other since then, and has had no further symptoms.

The first two patients had gallstones. The third patient was a man in his early forties who had had symptoms of many years' duration. Roentgenographic study showed a perfectly normal functioning gallbladder; upper and lower gastrointestinal studies were negative except for questionable duodenal dilatation. At surgery eight tiny gallstones less than 1 mm. in diameter were found. He had a definite compression of the fourth portion of the duodenum. I performed cholecystectomy and freed the duodenojejunal junction. His symptoms have been completely relieved.

Most of the cases I have seen since then have been of the type described by Dr. Rosenblatt, and they occur in the asthenic, thin patient in whom the symptoms are characterized by nausea and distress after eating, particularly an occasional "gurgling" and regurgitation of fluid in the upper abdomen. Occasionally they are relieved by rolling over on their abdomen.

I have collected records of twelve patients, eight of whom are mine, who were treated at Provident Hospital since 1954. I found none prior to that time. Two of the four whom I did not see personally were not operated; one was a sixty year old woman who had an associated ulcer, and the other was a twenty-six year old woman with a marked anxiety tension state. One patient, a sixty-five year old woman, underwent duodenojejunostomy for a huge dilated duodenum, with relief of symptoms. Another patient was a four year old mental defective from Alaska who had, in addition to the duodenal band, compression of the fourth portion of the duodenum.

One of my patients was a thirty year old woman who had a small duodenal ulcer. Roentgenograms suggested a compression of the fourth portion of the duodenum. I did not operate upon her, but this may have to be considered subsequently. I have not seen her recently.

Five of the other six have had gallbladder disease with cholelithiasis, and one had chronic cholecystitis without stones. One patient who did not have any gallbladder disease was a woman in her middle thirties who had had a uterine suspension. She then underwent vaginal hysterectomy, and prior to that time had had a bowel obstruction. She had a chronic pelvic inflammatory disease in the remaining ovary and stated that since childhood she had experienced nausea and vomiting every time she ate.

At operation I suspected lower abdominal adhesions, but I made my incision high enough to free the duodenojejunal junction. She has been absolutely free of symptoms since. I believe that this is a definite syndrome and that we should look for it more often.

MAX GASPAR (Long Beach, Calif.): I should like to point out a curious fact. We rarely see compression of the duodenum with abdominal aortic aneurysms even in those that arise above the renal arteries, and often the duodenum is flattened out on top of the aneurysm. At times the aneurysm

erodes into the posterior wall of the duodenum. However, I do not recall seeing this syndrome in a patient with an aneurysm.

Kaiser and Shumacker pointed out that the duodenum was compressed by the nutcracker that Dr. Jones depicted between the superior mesenteric artery and the aorta. From this consideration, it seems to me that Drs. Rosenblatt and Zeller have described a more important etiologic factor than the nutcracker one.

JOSEPH A. WEINBERG (Long Beach, Calif.): This subject merits thorough discussion. We are dealing with a symptom complex which is not clearly defined, and there is a great question about the possible benefits of surgical treatment. It may well be that the findings on roentgenographic examination are not related to the symptoms of which the patient complains. This way explain the frequent failure of operative procedures to relieve the syndrome. I realize that the authors have not implied that their operative procedure is for frequent use nor for use in every instance in which there is roentgenographic evidence of impairment of the duodenal lumen in its third portion. I only wish to emphasize that this type of procedure should not be used frequently for an entity which is so difficult to prove.

EARL P. LASHER, JR. (Seattle, Wash.): Some time ago Dr. Harkins' resident, Dr. Listerud, made a study of the abdominal crura of the diaphragm, and I asked him at that time if he would study something that has been known to anatomists for a long time concerning the wandering of bundles of striated muscles from the left or right crus well down into the ligament of Treitz and on to the duodenum. This is demonstrable in most normal people and it varies a great deal.

I wish to ask Dr. Jones whether he has biopsied any of these areas, and if so, if this anatomic observation could be confirmed. If it could be, it would account for variations in the symptomatology, and it would perhaps account for the increase of symptoms in people subjected to stress who might be thought of as carrying an additional tone in their striated muscle. The importance of direct pressure by the vessels might well be less than that of this muscle raising the duodenum toward the apex of the "U." It should also be noted that the left renal vein lies in this same crotch. Therefore, it would be only logical to expect an accompanying varicocele in all males afflicted.

ALLEN M. BOYDEN (Portland, Ore.): I wish to mention a recent experience I have had relative to this subject. I suspect that occasionally there is a patient with this mechanical problem but the indications for duodenojejunostomy must be infrequent.

Recently I had occasion to disjoin such an anastomosis in a young man who had been operated on for this some two years before. His symptoms before the anastomosis were compatible with the picture previously described by Dr. Jones. Asthenic and slender to begin with, the patient had promptly lost 20 pounds after the original operation. He noted that his symptoms were aggravated postoperatively and had asked his doctor to reoperate to restore the original situation.

We were fortunate to have had an upper gastrointestinal series performed some five years prior to surgical treatment. It had shown an irritable duodenum and was thought compatible with duodenitis.

Our exploration revealed no duodenal obstruction or other intra-abdominal pathologic condition. I hope that he will be able to regain his normal weight with simple dietary measures.

Not long before his death in 1933, Dr. Robert Coffey, one of the founders of this organization, realizing, I am sure, the fine contribution he had made in the treatment of carcinoma of the bladder by transplantation of the ureters into the large bowel, commented that the greatest operation he had developed was one known as the "hammock" operation for visceroptosis. However, this operation failed to gain favor. I suspect that many of his patients, often the same asthenic type, had symptoms similar to the group reported today.

LEONARD D. ROSENMAN (San Francisco, Calif.): I have had little experience with the anatomic nutcracker. I wish to discuss a surgical nutcracker at the fourth portion of the duodenum which was man-made.

The patient was a fifty year old woman who had carcinoma and multiple polyps in the left half of the colon. Left hemicolectomy was performed with anastomosis of the transverse colon to the upper rectum. About two weeks postoperatively she began to vomit and shortly thereafter roentgenographs illustrated the retention and dilatation of the duodenum one hour after she had taken the barium. Two and three hours later the picture was much the same.

A second operation demonstrated that cicatricial contracture of the root of the transverse mesocolon had compressed the duodenum in the same region that Dr. Jones described previously. It was relieved by duodenojejunostomy which was unfortunately over the top of the colon.

Since that time I have been careful when performing this operation to displace the entire small bowel to the right of the mesocolon and to release the ligament of Treitz so that the duodenum must not pass behind the transverse mesocolon.

S. AUSTIN JONES (closing): Dr. Farris' points concerning anesthesia are well taken. The majority of both patients and controls were given spinal anesthesia, but the exact data are not available.

As he points out, our incidence of positive roent-

genographic findings is singularly low. Recently we have found it of value to fluoroscope the patient leaning backwards 30 degrees from the vertical and push the stomach upward to allow a better view of the distal duodenum.

You will recall that of four of seven cases in which the diagnosis was missed, two were described as pyloric obstruction, one as an "atonic duodenum," and one as a "flabby duodenal wall." Of the remaining three with films showing no distention, the duodenum in one was bound down with adhesions following cholecystectomy. When freed it measured 10 cm. before and 15 cm. after air insufflation. The other two distended from 3.5 to 8.5 cm. and 5 to 11 cm. respectively. We can only conclude that this intermittent obstruction was not present in the latter two cases when the films were made.

As Dr. Farris mentioned, our cases were of the chronic type. In only one patient with a typical six-months' history, an acute obstruction developed of two days' duration. The patient was operated on for a supposed pyloric obstruction. This was a clinical error, as the patient was vomiting bile. The acute form is a real entity and may be a major postoperative problem if not recognized.

I do not believe that a Billroth II is a desirable procedure. The postoperative supine patient may have a complete obstruction, if only temporary, and the appreciable amount of bile and pancreatic juice building up behind this obstruction might cause real difficulty.

As previously mentioned by Dr. Strong, Dr. Rosenblatt's use of this procedure of mobilization of the duodenojejunal angle at such an early date, may be easier to perform and as effective as duodenojejunostomy.

Dr. Zeller's first biliary problem closely resembled one of ours. With reference to his second case, gastric dilatation has been incriminated, as he says, in pushing the viscera downward and aggravating a pre-existing condition.

Dr. Gaspar has brought up an interesting point regarding abdominal aneurysm. Most abdominal aneurysms are too far caudad to cause pressure at the apex of the nutcracker. There are a few important points that should be brought out in reference to anatomy. In the first place, the superior mesenteric artery does not pass downward directly anterior to the aorta, but rather down and to the right. Secondly, a duodenum may be V-shaped or U-shaped. In the V-shaped type the ascending limb frequently lies behind the superior mesenteric vessel and may be obstructed by it. The U-shaped duodenum is rarely obstructed by this mechanism. In 10 per cent of Dr. Pearsol's anatomic studies the duodenum did not even reach the right side of the aorta, so that this syndrome cannot develop anatomically. Hence, we see that the nutcracker analogy is only roughly true.

We included only the vascular obstructive cases in this study, and did not discuss the ones that had to do with a short ligament of Treitz. However, this is a definite entity.

The fact that too often these cases are handled surgically by the overenthusiastic physician is definitely true.

In conclusion, we hope that this preliminary report will stimulate some interest in this rare and rather controversial subject.

Replacements of the Esophagus

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METHODS to replace the esophagus have been explored from a variety of approaches. In the half-century since the first successful operation of this type was accomplished [*1*] many surgeons have investigated the problem. For a number of years skin tubes of various sorts were utilized to bridge the defect created by excision of the esophagus. Such tubes were fashioned from the skin of the anterior thorax and required multiple operative procedures to achieve a successful swallowing mechanism. These methods were mainly in use between World War I and the early 1930's. Scattered reports appeared during this time describing the use of segments of intestine to replace the esophagus. For a full quarter-century after the report of the first successful use of the large intestine, only twenty cases in which the colon was used as an esophageal transplant were reported; even fewer cases were on record describing the use of other intestinal segments [*2*].

César Roux [*1*] of Lausanne initiated the era of esophageal substitution in 1907. In that year Roux operated upon an eleven year old boy with esophageal stenosis resulting from a caustic injury. A segment of jejunum was passed subcutaneously into the neck to replace the damaged esophagus. However, the second stage of the operation, the anastomosis in the neck, was delayed for another four years.

Meanwhile, Herzen [*3*] of Moscow performed the first complete operation, using the jejunum as had Roux. Herzen's patient was a twenty year old woman with an intractable esophageal stenosis resulting from the ingestion of sulphuric acid solution six months previously. The three-stage procedure was completed over a period of three months. Initially, the segment of jejunum was transferred to an antethoracic position beneath the skin. One month later the distal end of the jejunum was anastomosed to the stomach and after another six weeks the cervical anastomosis was completed (1908).

The successes of Roux and Herzen stimulated others to attempt similar procedures. However, only four successful cases in which the jejunum was used were on record by 1915 [*4*]. In 1911 the German surgeon Georg Kelling [*5*] had performed a transplantation of the transverse colon as an esophageal substitute. The patient was a forty-three year old woman with an obstructing carcinoma of the esophagus 29 cm. from the incisor teeth. The transverse colon was transposed subcutaneously as high as the level of the nipple. Before a skin tube could be fashioned between the colon and esophagus the patient succumbed to the effects of the primary carcinoma. During the next twenty-five years only a handful of reports describing the use of the colon as an esophageal substitute were published [*2*].

Although suggestions for its use were made as early as 1913 [*6*] and again in 1920 [*7*] it was not until shortly before World War II that surgery of the esophagus was implemented by the development of procedures whereby the stomach was utilized to replace the esophagus [*8*]. With certain exceptions, the stomach as an esophageal replacement has withstood the test of time. To this day the stomach is used to replace the esophagus more often than are any other intestinal segments, especially in the management of esophageal cancer. Some dissatisfaction with the results of the use of the stomach in the treatment of benign diseases of the esophagus has led to a continuing search for more satisfactory methods of esophageal substitution [*4,9–12*]. The stomach, however, still has considerable value, even in the treatment of benign stenoses. Perhaps some of the dissatisfaction may be traced to the earliest use of the stomach as an esophageal replacement when

some of the aspects of the prevention of regurgitant esophagitis were not fully appreciated.

It is admitted that the stomach admirably satisfies the criteria for a suitable replacement of the esophagus. It has a plentiful and easily preserved blood supply which ensures its viability, a sufficient length to span the entire thorax into the neck if necessary, a satisfactory caliber throughout its entire extent, and it is resistant to the effects of peptic digestion. Except in those uncommon instances in which the gastric pouch is rendered useless by scarring, fibrotic contraction or other conditions, the stomach is readily available for use and can be mobilized without fear of vascular insufficiency. When the stomach is used the replacement procedure can be performed relatively easily, in contrast to operations in which other intestinal segments are used.

Replacement procedures using segments of intestine may prove too costly in terms of morbidity and mortality when compared to the results of the use of the more easily mobilized stomach. In children, however, in whom intestinal segments, especially those of the colon, are well suited for mobilization and substitution, and when the procedure is being performed for benign disease, such segments may be of greater use and success than in older age groups.

A review of the records of patients at the University of California Medical Center, San Francisco, who underwent esophageal resection and its replacement with the stomach shows that esophagitis will remain minimal or may even fail to develop if the resection of the esophagus is sufficiently extensive to allow the esophagogastrostomy to be placed high in the thorax or neck. If the anastomosis is performed at the level of the aortic arch or above, the development of a significant degree of esophagitis is uncommon. Most of the experience in this respect has been in the management of esophageal carcinoma. However, even in the treatment of benign esophageal disease, if the gastric pouch is elevated to a high position the presence or absence of esophagitis should be expected with equal frequency to those cases in which the esophagus is resected for carcinoma.

Early experience in the management of benign strictures of the lower esophagus by limited resection and esophagogastrostomy at a point low in the thorax was unsatisfactory because of the almost inevitable development of esophagitis in the proximal esophagus. Several mechanisms may account for this distressing result. In the first place, most of the stomach in these circumstances lies in its normal position, since only a small portion of it is elevated into the thorax. Most of the stomach remains in the abdomen and is therefore subjected to positive pressure while the intrathoracic portion of the stomach is in an area of negative pressure. (Fig. 1A.) This difference in pressure enhances retrograde flow of juices, which is increased further by the absence of a sphincteric mechanism at the esophagogastric anastomosis. Secondly, a pyloroplasty was not often performed as a part of the procedure in the early management of this problem. Whether or not a pyloroplasty is performed, differential pressure relationships may still promote regurgitation.

However, when the stomach is placed high in the thorax after resection of most of the esophagus, the gastric pouch is essentially an intrathoracic organ, sustaining only negati[redacted]essure throughout its entire course. (Fig. 1B.) Furthermore, high placement of the stomach in a vertical position allows for gravity flow of gastric juices away from the area of the anastomosis. This is true even when the patient is in the recumbent position while sleeping, since the slightly elevated position of the head allows proper flow or at least minimal regurgitation during the nighttime hours. In either event, a properly performed pyloroplasty now stationed at the level of the esophageal hiatus of the diaphragm militates against improper flow of juices. The effect of vagotomy, by reducing gastric secretion, is an additional benefit when the stomach is used. The vagus nerves may or may not be resected when other replacement or bypass procedures are used.

Although the stomach is ideally suited for an esophageal replacement because of its caliber, length, blood supply and ease of mobility, several objections to its use arise on clinical as well as anatomic grounds. Occasionally, distention of the intrathoracic gastric pouch after eating produces distress. Slow emptying of the stomach related to a poorly functioning pyloroplasty may play a role in the development of these symptoms. In our experience, however, this type of distress, although relatively common, is annoying rather than disabling. Patients may develop a type of dumping syndrome which varies considerably in severity. Such distress has been noted with almost equal fre-

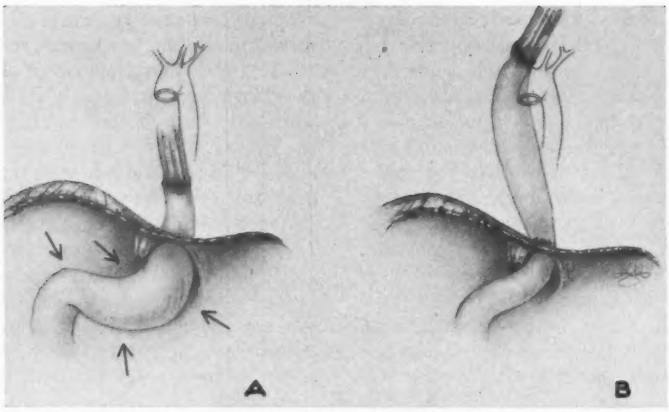


FIG. 1. A, the positive pressure upon the abdominal portion of the stomach enhances the flow of gastric juice upward; B, when the entire stomach is intrathoracic it is not subjected to differential pressure relationships.

quency and severity when other intestinal segments are used as esophageal substitutes [12]. Nutritional problems exist to a certain extent regardless of the type of esophageal substitute. For the most part, the reconstitution of a swallowing mechanism of adequate caliber contrasts so markedly with the preoperative obstructive state that a reasonably satisfactory nutritional status is usually obtained.

Certain anatomic objections to the use of the stomach exist. These relate to the use of the stomach as an esophageal replacement when the esophagus is excised for malignant disease. It is well known that esophageal carcinoma may extend for a considerable distance submucosally beyond the palpable limits of the lesion. The stomach shares by continuity a common lymphatic pool with the esophagus. However, most of the downward lymphatic drainage passes extragastrically, rather than submucosally, into the celiac nodes. In spite of this pathway the stomach lies in such close proximity that retrograde flow from the lymph nodes may involve the lymphatics of the stomach by direct continuity. By using the stomach as an esophageal replacement in the management of esophageal carcinoma, a pool of actually or potentially invaded lymphatics may be transposed into the thorax. From a clinical standpoint, however, it is fortunate that the stomach usually remains free from lymphatic involvement in spite of these anatomic considerations except when the primary lesion is located at a point close to the stomach. Autopsies of patients treated for esophageal carcinoma show that recurrent malignancy in the gastric pouch is uncommon. On the contrary, recurrent disease is most often extragastric in location.

In spite of the advantages in using the stomach as an esophageal substitute, some dissatisfaction, especially when it is used in the management of benign esophageal disease, has stimulated the search for more satisfactory substitutes. The jejunum and colon have been utilized but not without certain limitations and disadvantages.

The jejunum is a satisfactory substitute for the esophagus and has been used with success by a number of surgeons throughout the world. Yudin [4] has reported the greatest number of procedures using the jejunum, and his experience includes more than three hundred patients. The limitations to the use of the jejunum are largely anatomic. The short vascular arcades may limit the length of jejunum available for replacement. In this respect, necrosis of the distal portion of the jejunal transplant causes most of the difficulties associated with its use. In the management of benign strictures limited to the lower esophagus, long segments of jejunum are unnecessary so that marginal necrosis in these instances is not a problem. When the entire thorax must be spanned, however, no little concern exists about the blood supply at its distal extremity. That this is an important consideration is supported by the fact that the anastomosis of the jejunum to the cervical esophagus is almost always delayed for

a few days in order to observe the vitality of the transplant. In a recent report [*13*] which describes the experience with fifty-five jejunal transplants into the neck, necrosis of all or part of the lengthy jejunal substitute occurred in ten patients, and three of these patients died.

Other limitations to the use of the jejunum as an esophageal substitute are based upon physiologic considerations and the long term observations of patients undergoing these procedures. The jejunum as well as any other transposed intestinal segment maintains peristaltic activity in its new habitat and will perform admirably as a food conduit. The jejunum, however, is highly susceptible to the effects of peptic activity. When it is anastomosed to the stomach as the completed lower end of the transplant, the jejunum is subjected to the relentless effects of peptic digestion. Without the benefit of a valve mechanism at this anastomosis some degree of jejunitis, esophagitis, or both, may be expected. This is probably true, although to a lesser extent, even if an adequately functioning pyloroplasty exists. It is said that the jejunum may act as its own valve mechanism by virture of its peristaltic activity. Others have shown that for some unknown reason the jejunal mucosa in its new position may exhibit a greater resistance to peptic digestion than either the stomach or the duodenum [*14*].

Our limited clinical experience with the use of the jejunum compares favorably with the reported results of others [*14,15*]. However, our more extensive experience in the animal laboratory has been less satisfactory. As a long term experiment segments of jejunum measuring 25 cm. in length were inserted in twelve dogs between the transected thoracic esophagus and the fundus of the stomach as previously described [*11*]. A pyloroplasty was added to each procedure. The project assessed over a long period the ability of the transposed jejunum to act not only as a conduit for the passage of food but also its ability to prevent the development of esophagitis. Animals so prepared were observed for one to three years. Although the nutritional status of the animals was maintained quite satisfactorily, a steady and progressive downward trend in their weight curves was noted. The animals were examined by esophagoscopy at intervals up to three years after the initial operation. Varying degrees of narrowing, edema and inflammation were noted, not only at and above the jejunoesophageal anastomosis but also in the transposed segment of the jejunum.

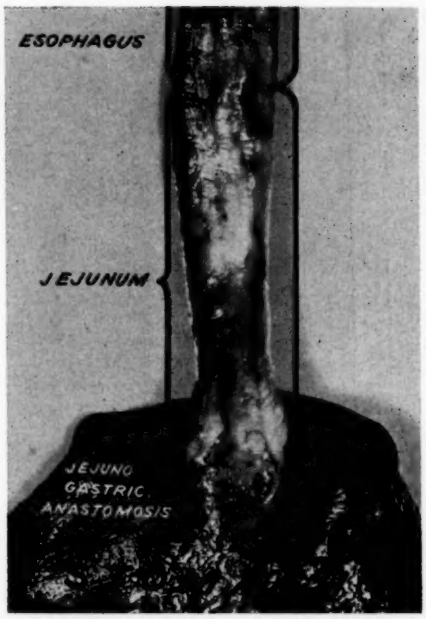


FIG. 2. Autopsy specimen of a dog in which replacement of the lower esophagus was performed twenty-eight months previously. The intact specimen has been turned so that the mucosal surface is exposed. The jejunal segment (bracket) is inflamed. Moderate esophagitis exists immediately proximal to the esophagojejunostomy. At the lower end of the specimen the stomach shares in the inflammatory process. Note the fibrinous plaques (white) scattered throughout the length of the specimen.

On esophagoscopic examination, a moderate esophagitis was uniformly seen in the area immediately proximal to the jejunoesophageal anastomosis. A greater degree of inflammation was usually noted in the jejunal segment. No ulcer craters were demonstrated in the area of the esophagus, jejunal segment or stomach, even in those animals observed for a full three years. Postmortem studies verified the presence of nonspecific jejunitis as well as esophagitis, both grossly and microscopically. (Fig. 2.) It appeared from these long term observations that the segment of jejunum acted not so much as a valve protecting against regurgitation of gastric juices but rather as a long conduit over whose length the juices found some difficulty in progressing [*16*].

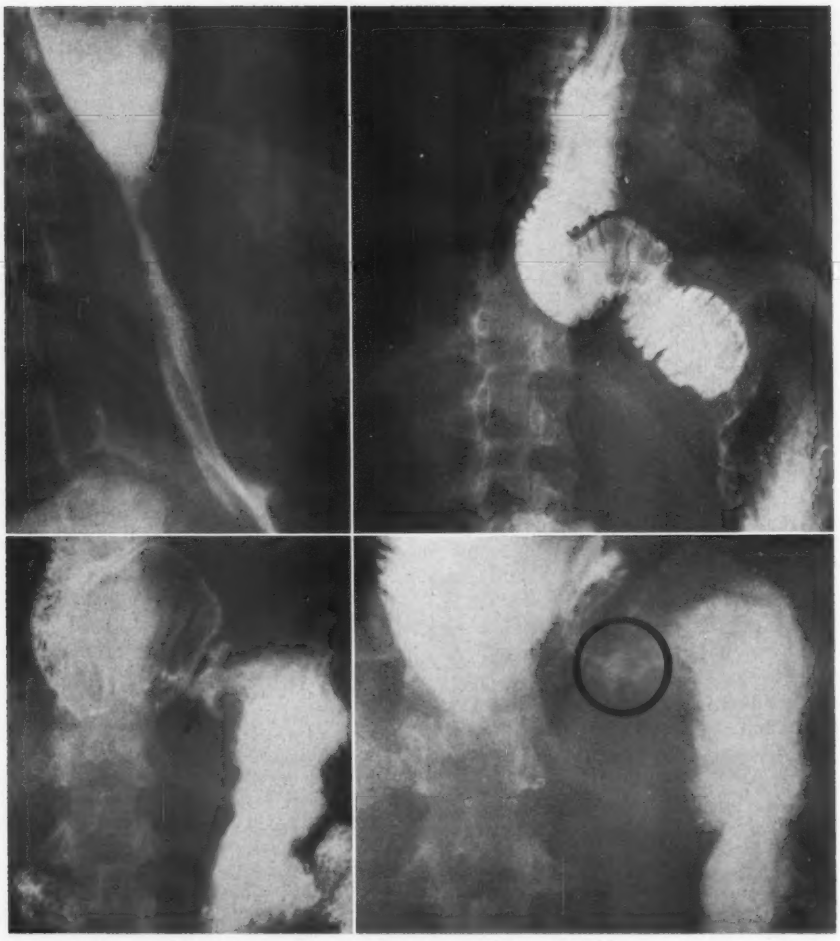


FIG. 3. A, the acquired short esophagus with stricture and proximal dilatation is evident. B, the stenotic lower esophagus has been replaced by a transposed segment of jejunum. C, four years later a persistent irregularity (arrow) could be demonstrated at the esophagogastric junction. D, after a second bout of upper gastrointestinal bleeding an ulcer crater was identified (circle).

The results in three patients who underwent procedures in which segments of jejunum were used to replace diseased segments of the lower esophagus have been satisfactory in two cases but less so in the third. In all the patients the procedure was performed for intractable benign stenosis of the lower esophagus. The untoward developments occurred in a thirty-one year old woman with intractable stenosis of the lower esophagus resulting from shortening and stricture due to a hiatal hernia, possibly congenital (Fig. 3A.) Previous treatment had been only partially satisfactory. A long segment of jejunum was inserted between the esophagus and the fundus of the stomach after excision of the stenotic scarred area in July 1955 (Fig. 3B). A bilateral vagotomy and a Heineke-Mikulicz pyloroplasty were performed.

The procedure was relatively successful from a symptomatic standpoint although the patient had frequent dysphagia and epigastric distress. Several roentgenologic studies for peptic ulcer were negative in spite of symptoms suggestive of ulceration in or near the stomach. In February 1959, nearly four years after the jejunal replacement procedure, massive upper intes-

tinal hemorrhage occurred. Roentgenologic studies at that time showed a persistent irregularity at the jejunogastric junction (Fig. 3C). Esophagoscopic examination showed that the lower esophagus near the esophagojejunal anastomosis was fiery red and edematous. The jejunal segment was also inflamed but to a lesser extent. Further downward at the gastrojejunal anastomosis the mucosa was severely inflamed. No ulcer crater could be seen. Massive hemorrhage occurred again in May 1959, while the patient was restricted to an ulcer diet. Roentgenologic studies at that time showed considerable reflux of gastric juices into the jejunal segment and esophagus, and a large ulcer crater was demonstrated at the jejunogastric junction (Fig. 3D).

This limited clinical experience is not sufficient to justify conclusions regarding the use of an isolated segment of jejunum to replace the esophagus. These and other reported clinical results cannot be regarded as altogether displeasing. It is suggested, however, that the longer term and more extensive experimental evidence suggests that certain difficulties, both technical and clinical, may arise in the use of an isolated jejunal segment. This is not to decry its use but rather to point out its inherent limitations.

Other investigations in the laboratory showed that a naturally occurring intestinal valve could be substituted for the cardioesophageal mechanism. In studies on twenty-four animals it was demonstrated that an ileocolic segment, consisting of 25 cm. of terminal ileum, the ileocolic valve and 5 to 7 cm. of ascending colon, could be transposed to replace the lower esophagus and upper stomach (Figs. 4 A, B and C). Pressure studies at autopsy showed that the ileocolic valve remained competent in most instances until the intragastric pressures exceeded 50 cm. of water [17]. Studies in which injections of histamine were used demonstrated that the transposed colonic segment, as well as the ileum, were more resistant to peptic digestion than the stomach or duodenum. After prolonged stimulation deep ulcers, some of which were perforating, could be seen in the stomach or duodenum, but the ileocolic segment, especially its ileal portion, remained free from significant inflammation. (Fig. 5.)

Based on these observations in the laboratory, the ileocolic-valve replacement procedure was undertaken in five patients who had intractable benign stenosis of the lower esophagus. Severe dysphagia was the predominant symptom in each patient. Stenotic areas were demonstrated roentgenologically in the lower esophagus along with proximal distention. A satisfactory result has been obtained in four of the five patients who were treated in this fashion. The fifth patient, who had bilateral fibrotic lung disease in addition to intractable stenosis of the lower esophagus, died in the immediate postoperative period. In this instance the procedure was ill chosen in view of the patient's condition. The other four patients operated upon since this initial experience have obtained a satisfactory swallowing mechanism and have a positive nutritional balance. The longest period which has elapsed since the first successful procedure of this type is four and a half years.

A representative patient in this group was a forty-six year old woman who had intractable

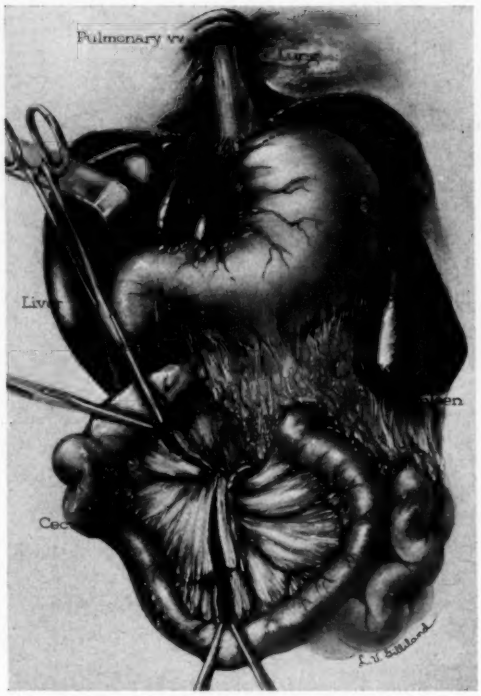
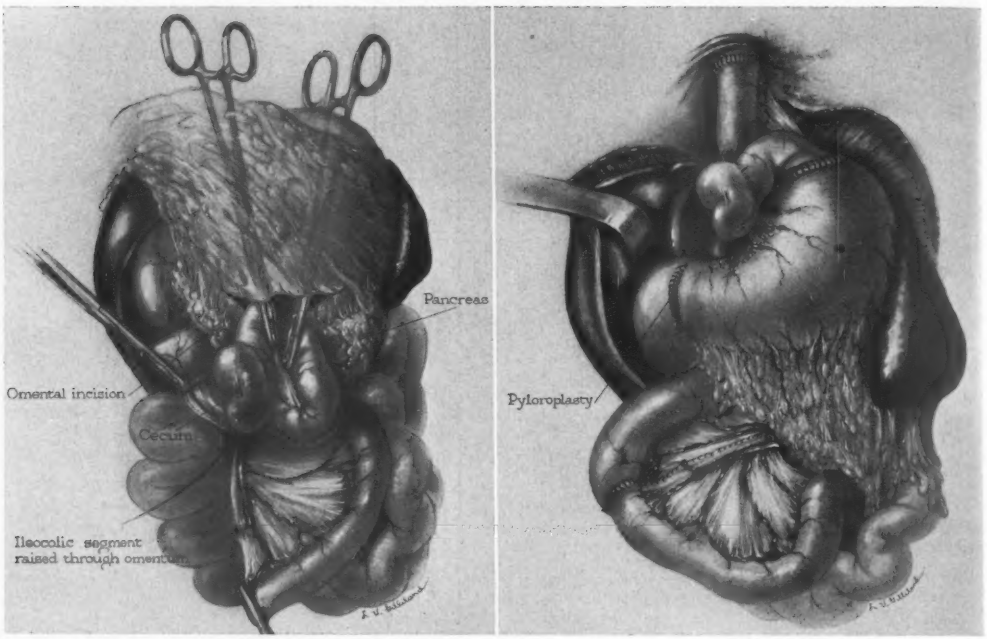


FIG. 4A. The ileocolic valve substitution procedure in the experimental animal. The ileocolic segment is isolated with preservation of the ileocolic vessels, and the dotted lines indicate the extent of excision.



4B 4C

FIG. 4. The ileocolic valve substitution procedure in the experimental animal. B, the ileocolic segment is transposed upward through the lesser sac behind the stomach. C, the ileocolon lies in the left upper abdomen replacing the excised eophagus and stomach, and the pyloroplasty and end-to-end ileocolostomy complete the procedure.

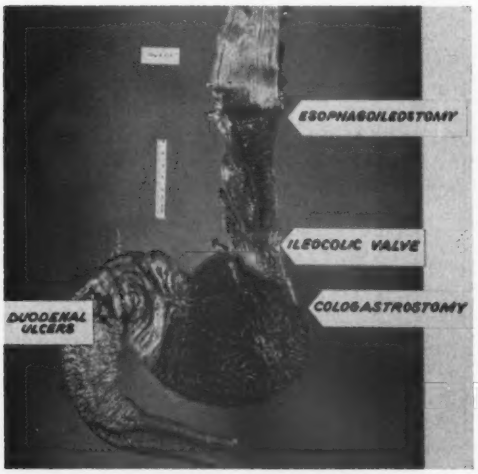


FIG. 5. Autopsy specimen of a dog in which excision of the lower esophagus was performed five months previously. Replacement was by means of the ileocolon. After prolonged stimulation by injection of a histamine preparation, perforating ulcers can be seen in the duodenum, surrounded by an intense inflammatory reaction. The stomach, ileocolon and esophagus remain relatively free from inflammation.

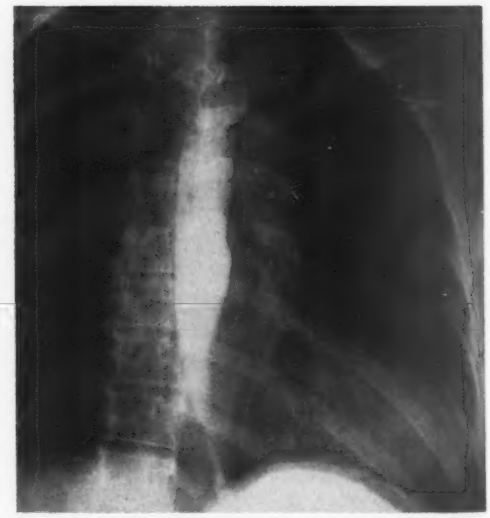
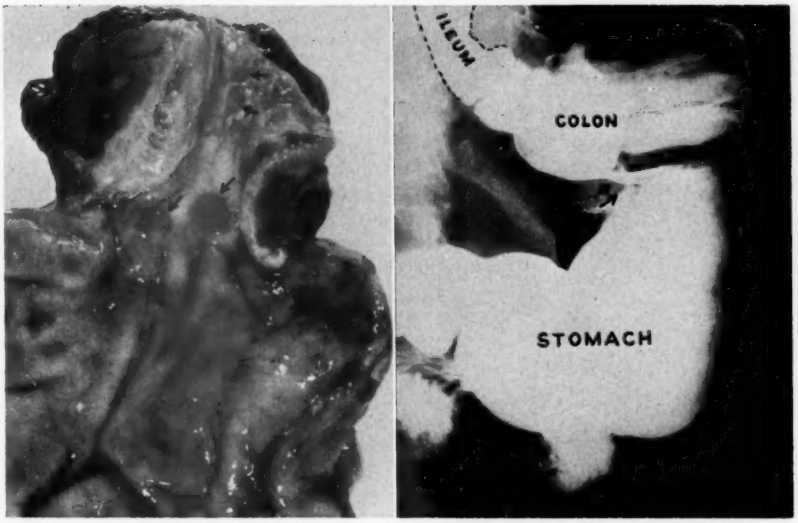


FIG. 6A. The stricture in the lower esophagus was resistant to dilatation by instrumentation.



6B 6C

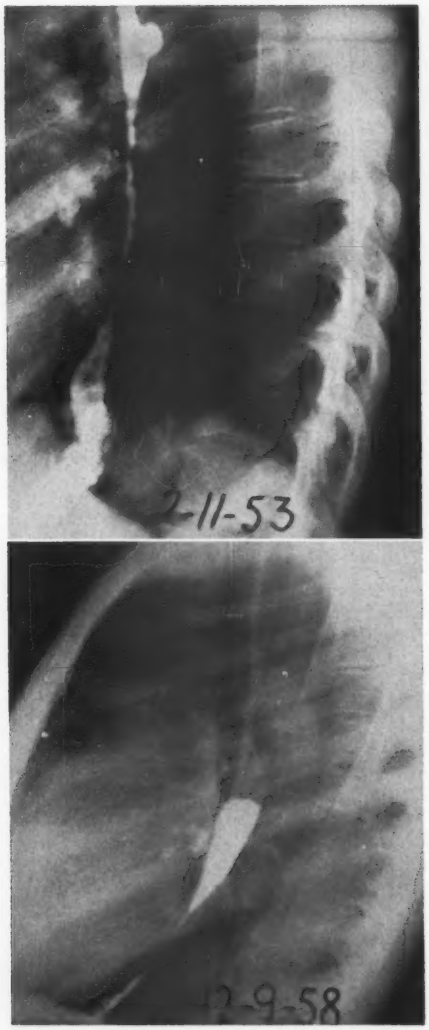
FIG. 6. B, the excised area shows narrowing, intramural fibrosis and four ulcer craters (arrows) in the lower esophagus. C, postoperative roentgenologic studies show a functional ileocolic segment transposed between the lower esophagus and upper stomach (arrow at ileocecal valve and double arrow at cologastric anastomosis).

stenosis of the esophagus due to reflux esophagitis (Fig. 6A). Since conservative maneuvers were unsuccessful, the strictured area in the lower esophagus was resected and replaced by an ileocolic segment. The pathologic specimen showed not only extensive scarring intramurally but also an intense inflammatory process in the mucosa, including multiple deep ulcerations (Fig. 6B). The patient has remained free from distress and is able to swallow satisfactorily. A roentgenologic study of the upper gastrointestinal area shows a well functioning upper intestinal unit (Fig. 6C).

Among twelve patients in whom the entire right colon was utilized to bypass the thoracic esophagus, five had benign strictures involving all or most of the esophagus as a result of accidental or deliberate ingestion of caustic substances. Bypass of the stenotic esophagus in each of these five patients with benign disease has been successful and an adequate swallowing mechanism has been achieved. None of these patients, however, is completely free from distress when eating. Complaints vary from minor sensations of fullness in the neck, palpitations of the heart, and occasional foul breath, to the most distressing symptom which appears to be a well developed dumping syndrome that may be related to the pyloroplasty performed as part of the procedure.

The one patient who is nearly free from postoperative symptoms is a forty-three year old Negress who had a long stricture of the esophagus as the result of accidental ingestion of a caustic solution in 1951 (Fig. 7A). Over a period of several years as many as thirteen antegrade dilatations were performed which provided partial but temporary relief. The patient was restricted to a liquid diet for seven years. Dysphagia became progressively more severe so that virtually complete obstruction was present by December 1958 (Fig. 7B). A gastrostomy was established to provide retrograde dilatations. The stenosis was so severe, however, that instruments could not be passed upward. In January 1959 the stenotic scarred esophagus was bypassed, using the right colon inserted into the neck through a substernal tunnel. (Fig. 8.) The patient is well satisfied with her present ability to swallow, thirteen months after the operation (Fig. 9A and B).

Of the seven patients who had right colon esophageal bypass procedures performed for malignancies, six had carcinoma of the midthoracic esophagus and one had an extensive adenocarcinoma of the fundus of the stomach



7A

7B

FIG. 7. A, two years after the caustic injury a long area of stenosis of the esophagus was still present. B, some improvement was noted in the upper esophagus but by December 1958, virtually complete obstruction of the lower esophagus had developed.

with involvement of the lower esophagus. All had complete or nearly complete obstruction. In some cases the patient's saliva could not be swallowed. Most of the patients were thin, partially emaciated, and had been able to tolerate only liquids for periods of weeks or months prior to admission to the hospital.

In only one instance was the technical procedure a failure. This occurred in a sixty-five year old man who had a mid-thoracic esophageal carcinoma and who was still obese at the time the definitive diagnosis was made. An attempt to transpose the right colon through a substernal tunnel to the cervical esophagus was unsuccessful. The entire segment became gangrenous because of extensive thrombosis of the venous drainage system of the right colon during the process of mobilization. An esophagostomy and gastrostomy were established. The patient's condition deteriorated rapidly in the immediate postoperative period and he expired on the fifth postoperative day. An autopsy revealed extensive purulent mediastinitis as well as widespread necrosis of the liver.

Another elderly patient expired suddenly in the fourth week after a successful right colon bypass procedure had been accomplished. An autopsy showed that death was due to aspiration of what appeared to be gastric contents.

In none of the patients operated upon for malignant disease, in which the colon bypass procedure was used, was a second planned operation for the removal of the primary esophageal growth ever possible. In each instance the general condition of the patient did not justify the risk of removal of the primary growth after it had been bypassed by the initial procedure. However, four patients received radiation therapy for the esophageal lesions. The patient who had extensive fundic carcinoma involving the lower esophagus was the most severely obstructed prior to operative treatment. In this case the bypass procedure was admittedly a palliative one. The swallowing mechanism functioned well during the remaining ten months of life. Death was due to exsanguination from the primary gastric carcinoma.

During the same five-year period in which seven patients with malignancy of the esophagus were managed by means of a right colonic substitution, forty-nine other patients were treated for cancer of the esophagus by a variety of surgical procedures. Thirty-one of these patients underwent resection with restoration of continuity by an esophagogastrostomy. Some of the advanced lesions were treated conservatively by a gastrostomy for feeding purposes followed by irradiation, nasogastric tube for feeding plus irradiation, and in a few instances by tube feeding alone. The patients receiving the right colon substitution procedure during this same period were, therefore, a selected series whose management and results could be compared with the patients treated by other

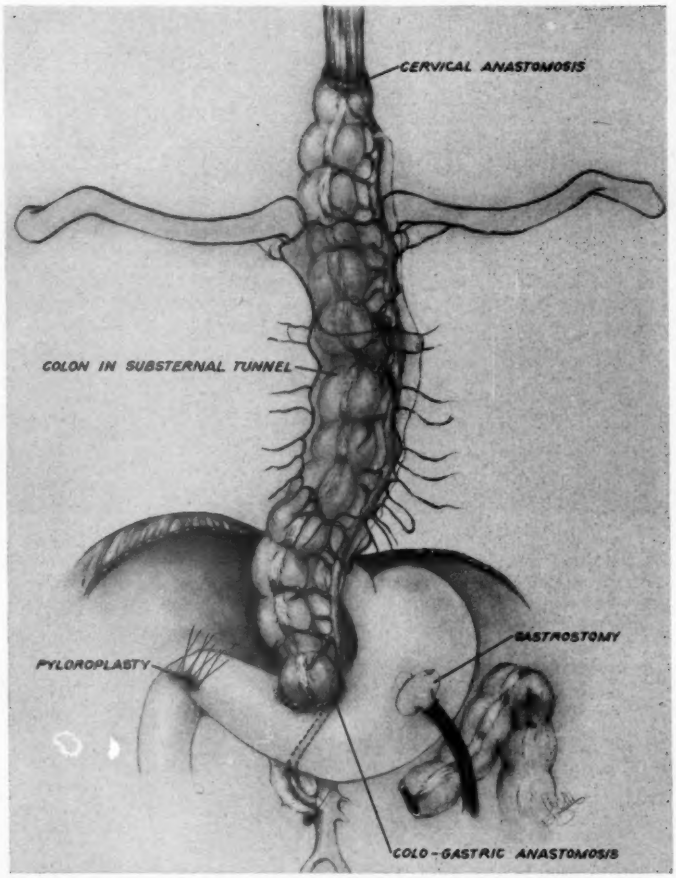


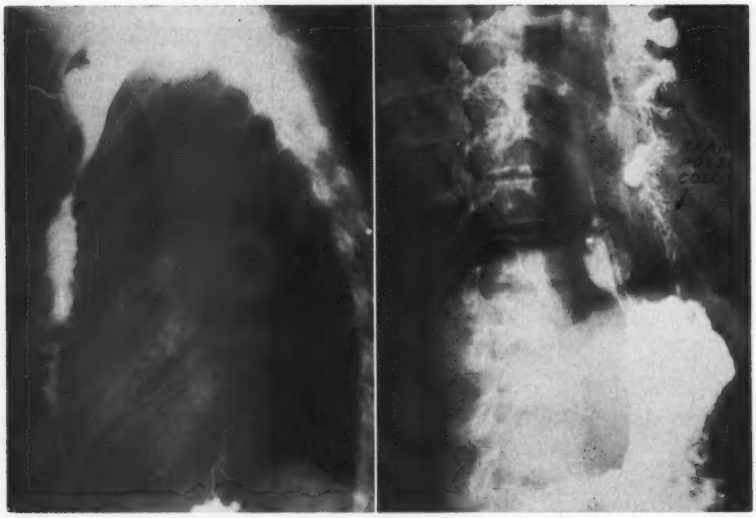
FIG. 8. The entire thoracic esophagus is bypassed by the use of the right colon inserted through a substernal tunnel with anastomosis to the esophagus in the neck.

TABLE I
ESOPHAGEAL REPLACEMENTS
University of California, San Francisco
Five-Year Period 1955–1959

Segment Utilized	Total Number	Benign	Malignant
Jejunum	3	3	0
Ileocolon	5	5	0
Right colon	12	5	7
Transverse colon	2	2	0
Other (stomach 31)	49	0	49
Total	71	15	56

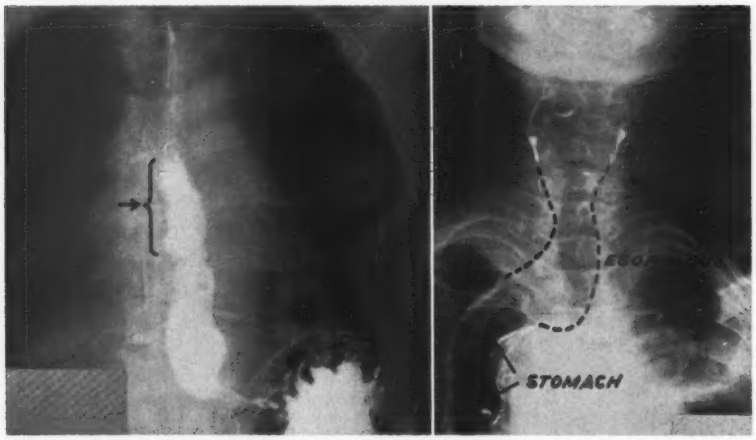
methods. A statistical analysis is presented in Table I.

Although the number of patients in each group discussed here varied considerably so that numerical comparisons are impossible, certain clinical impressions warrant discussion. In the first place the use of the stomach to restore gastrointestinal continuity after esophageal resection for malignant disease has proved to be satisfactory in the experience of most surgeons interested in the management of these problems. Esophagitis, although usually present in varying degrees of severity, has not been a significant postoperative problem as long as the stomach can be brought to a sufficiently high position in the thorax. (Fig. 10.) Moreover, a procedure in which it is planned in advance to



9A 9B

FIG. 9. A, the lateral roentgenogram shows the well functioning transposed right colon in the substernal tunnel. On the anterior-posterior film note the regurgitation into the esophagus which remains in its anatomic position. B, a hiatal hernia has been produced by shortening of the esophagus from the effects of the original injury.



10A 10B

FIG. 10. A, the carcinoma in the midthoracic esophagus had produced dysphagia for five months. B, gastrointestinal continuity was recreated by esophagogastrostomy high in the thorax. The patient has remained free from esophagitis for six years.

use the stomach to replace the esophagus infers that the esophageal cancer itself will be exposed and resected in the majority of cases. If it is not resectable the primary growth can be palliatively bypassed at the same procedure. In none of the cases in our experience with the use of right colon replacements for esophageal cancer was the primary growth resected, nor was it ever directly explored. Rather, radiation of the primary growth was undertaken since the slow recovery of the patient from the bypass procedure did not warrant a major thoracotomy at a later date. Similar experience in the treatment of esophageal cancer by this method has been noted by others [*12*].

The question may be raised whether the

major substitution procedure is justified in the management of esophageal malignancies in the absence of complete obstruction to saliva, since a feeding gastrostomy followed by radiation therapy accomplishes essentially the same end result with much less total trauma to severely debilitated patients. In our experience the creation of a gastrostomy for feeding purposes has been satisfactory as long as radiation in therapeutic doses was administered to the primary growth. Gastrostomy performed merely to prolong life without treatment being afforded to the primary growth is quite unsatisfactory. One wonders whether the major procedure of segmental intestinal transposition to connect the mouth with the stomach is justifiable, since a simple opening in the stomach accomplishes much the same effect.

It is in the management of benign stenoses of the esophagus that colonic segments should find their greatest use. Here the ravaging effects of relentless malignancy do not exist. The unrestricted span of life following the successful re-establishment of a swallowing mechanism by a colonic transplant justifies the major surgical procedure. Moreover, a second major operation is not required since the diseased esophagus may remain within the thorax without significant hazard.

Beyond these considerations the colon is admirably adapted for use as an esophageal substitute. It is long enough to span the entire thorax into the neck if necessary. In each of the twelve patients in whom the right colon was used, the segment was deliberately brought up into the neck for anastomosis to the cervical esophagus. This was accomplished even though the anastomosis might have been performed within the thorax in several instances, since a portion of the upper thoracic esophagus was normal. The anastomosis in the neck is easily performed and the passage of the colon through the substernal tunnel allows the procedure to be accomplished without opening the thorax. Furthermore, should leakage occur at the anastomosis, drainage into the neck is well tolerated, while leakage into the thorax may prove disastrous.

A shorter length of colon is needed when it is passed through the substernal tunnel into the neck than when it is transposed over the longer distance of the posterior mediastinum within the thorax. In children in whom the transverse mesocolon and blood vessels, as well as the colon itself, are long and often redundant, the use of the colon has been attended with the most satisfactory results [*18*]. A recent publication [*13*] from Eastern Europe reviews a series of seventy-eight operations to replace the esophagus by means of a loop of jejunum in fifty-five patients and with the colon in twenty-five patients. The results, complications and failures roughly parallel those of other authors in this country and elsewhere.

SUMMARY

The esophagus may be replaced by various means. The skin tubes fashioned from the anterior thorax which were formerly used to restore continuity after esophageal resection have been superseded by more direct methods. Resection of the esophagus for both malignant and benign disease has become a commonplace procedure. The ability to surgically remove many types of esophageal disease is well established. Some uncertainty still exists about the best method of restoring gastrointestinal continuity after esophageal resection.

The stomach is admirably suited for use as an esophageal substitute. It is easily mobilized and has attributes which make its use favorable from most aspects. Its shortcomings are minimal even when it is used in the treatment of benign disease. Experience has shown that if the esophageal resection is extensive and the esophagogastrostomy is performed at a high level in the thorax, esophagitis is not a frequent development.

The jejunum and colon are often used as esophageal replacements. The jejunum has certain limitations related both to its anatomically short vascular arcades and to certain physiologic and long term observations of patients undergoing these replacement procedures. The use of the colon presents fewer technical difficulties than does the jejunum, and is well suited for total or nearly total replacement of the esophagus. A discussion is presented here which questions the justification of the use of the colon or jejunum in the management of malignant disease of the esophagus. It is suggested instead that the stomach is a more satisfactory esophageal substitute in these instances. In the treatment of benign esophageal stenoses the colon or jejunum should find their greatest use as esophageal replacements.

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DISCUSSION

Lyman A. Brewer, III (Los Angeles, Calif): Although the last word has not been written on the technic of esophageal replacements, great progress has been made and this review of experience has been most stimulating to all of us who are interested in this subject.

I should like to discuss this entity from two points of view; first, benign lesions; and second, malignant lesions. We agree that colon is the ideal substitute for extensive replacement of the obstructed esophagus in benign cases. This is particularly true in children in whom a long, redundant mesentery is present, so that even cervical anastomosis can be done without tension or loss of blood supply. Both stomach and jejunum are much less satisfactory in our experience. Most patients with benign obstruction have had a previous gastrostomy which has to be closed to mobilize the stomach. This results in further limitation in size of the usually contracted stomach. Thus high cervical anastomosis is not feasible. The variations in the arcuate branches of the jejunum makes it unsatisfactory for high mobilization. Colon is a good substitute for the esophagus because of its blood supply, ease of sterilization with neomycin, and its resistance to peptic acid digestion which is most likely due to profuse mucous secretions. Studies by Ivy and Wangensteen have demonstrated this.

Doctor Grimes has reported their use of cecum for esophageal replacement in low benign lesions. The cecum is so implanted that the ileocecal valve serves as the cardiac sphincter. This technic is ingenious and we will be interested in its further clinical use.

In malignant lesions we have used the stomach very satisfactorily for esophageal replacement. There are several technical measures which we have employed which are important to the success of the operation. The angle of insertion of the esophagus into the stomach aids in prevention of esophageal regurgitation. This is a controversial subject and Ellis and others have presented conflicting evidence. However, the acute angle of insertion of the esophagus to the stomach allows the fundus to ride above the anastomosis and thus places tension upon it. To further provide a valve-like mechanism, the stomach is wrapped half way around the lumen of the esophagus. Watkins has recently reported the wrapping of the stomach around 90 per cent of the lumen of the esophagus to effect a valve-like mechanism. We believe that covering 50 per cent of the esophagus is sufficient and has minimized esophagitis.

When the stomach is brought up into the chest the phrenic nerve and function of the diaphragm should be preserved. One technic presented to this Society in 1948 consists of a curved incision of the diaphragm, saving the main phrenic nerve and the two main branches. Quite good diaphragmatic motion follows this technic, not only aiding in pulmonary function but also providing a pumping action on the stomach which may enhance gastric emptying. The final technical point is the performance of a generous Heineke-Mikulicz type of pyloroplasty in place of the myotomy that we formerly recommended.

The Berman tube has evoked a great deal of interest in this country during the past five or six years. We have had very poor results with it. Its use is based on the principle that there must be an airtight sealing of the tissues about the tube or a leak will occur. When you have cleaned out the mediastinum there is practically no tissue remaining to provide an effective seal. The mediastinal pleura is often extremely fragile in bypass procedures so that the airtight sealing cannot be achieved and the procedure is then doomed to failure.

We have continued to use hypothermia for patients with carcinoma of the esophagus who are poor risks for surgery and believe it has a very definite place in this type of operation. In the original series presented before this group two years ago, we had a mortality of three out of the first seven cases. In the last thirteen cases, we have lost only one patient operated upon with the aid of hypothermia. This improvement is due partly to improved use of this technic, but mainly to the fact that we are not operating upon patients for whom there are prohibitive risks. Blood loss must be accurately replaced as the hypothermic patient bleeds more than the normothermic one and tolerates hemorrhage poorly. One must constantly keep in mind the danger of secondary shock occurring from twenty-four to thirty-six hours after surgery. Preparations for blood replacement, and administration of vasopressors and corticoids should be made.

Time permits only a brief word about the approach to carcinoma of the esophagus. There is no question that certain cases have been reported with long term palliation following deep x-ray therapy. However, in our experience, when intensive deep x-ray therapy with 2,000,000 volt machines and rotational technics has been used the results have been disappointing. In advanced cases carcinomatous obstruction has been relieved only to be followed by cicatricial stricture. Gastrostomy feeding is necessary and the patient is not very happy with his inability to swallow food. Therefore, although it is impossible to present all the supporting evidence in this discussion, we believe it is worthwhile to perform palliative resection providing the technics employed result in the patient's ability to eat satisfactorily.

JOSEPH W. NADAL (Portland, Ore.): I am particularly interested in one aspect of the problem Dr. Grimes discussed. I was struck by the fact that one of his best results was in the patient who had a colon substitution retrosternally.

Six and a half years ago I operated upon an infant girl who had an esophageal atresia and a tracheoesophageal fistula. The distal esophageal segment was too short to permit an end-to-end anastomosis, so I performed a cervical esophagostomy and a gastrostomy for feeding. Having heard of Sherman's work and experiences with the use of the colon retrosternally, I employed this method of joining the space between the cervical esophagus and the stomach two years after the initial operation.

There is much to be learned about the long term success of this procedure. The questions have been raised as to whether children with this problem will be normal as they grow—whether they will be able to swallow normally, whether they will develop normally, and whether anemia and other complications will occur.

I saw this little girl recently. She has been entirely normal for a period of four and a half years since the colon substitution. She can eat anything. She goes to school. She has no anemia, and she never vomits.

ARTHUR J. HUNNICUTT (Oakland, Calif.): I would like to make a few comments on just one phase of this paper—the anastomosis of the esophagus to the transplanted portion of the bowel.

In all but one of the last sixteen patients in whom I have performed right ileocolon replacement of the entire stomach I have preferred a telescoping anastomosis between the esophagus and the terminal ileum, and so far I have no reason for not continuing it. The one patient who had an end-to-end anastomosis is the only one who has had to have dilatation (twice) for stricture.

The mucosa of the severed esophagus is turned up as a cuff and sutured on the outer wall of the esophagus. The cuff is turned up for a distance of about 1 cm. and held in place with four sutures of fine silk.

The seromuscular layer of the transected ileum is then sutured to the outer wall of the esophagus adjacent to the upper edge of the mucosal cuff. Thus, the esophagus is telescoped into the lumen of the bowel for a distance of 1 cm. Twenty-four to thirty sutures of fine silk are used in this one layer approximation.

In using this method I was trying to avoid the problem of having the line of anastomosis within the lumen. There is less danger of leakage with the scar relatively outside the lumen, and the scar is dilated not irritated by swallowed food. There has been no problem due to any differences in the size of the lumena of the esophagus and ileum.

I have preferred this telescoping anastomosis in small and large bowel resections for about twelve years. It was my satisfaction with this that led to its application to esophageal anastomoses.

E. N. SNYDER, JR. (Pasadena, Calif.): Some years ago Dr. Mathewson and Dr. Cohn reported favorably on their experience with the combined right chest and abdominal approach for esophageal lesions. I read this while I was still a resident and was allowed to carry out some esophageal resections using this technic. Dr. Gaspar and I reported our

experience eight years ago and we are just as enthused today with the advantages of this technic as we were then. The advantages referred to are better exposure with easier dissection and therefore, better cancer surgery, as well as minimal disturbances of cardiorespiratory physiology.

Drs. Mikkelsen, Rogers and I subsequently reported our experience with what we called "the two-team simultaneous thoracoabdominal approach to esophageal surgery." Others might refer to this as synchronous thoracoabdominal surgery. While this approach does have some limitations, it significantly reduces the operating time without increasing morbidity and without sacrificing the exposure and extent of resection inherent in the right thoracoabdominal approach.

H. Brodie Stephens (San Francisco, Calif.): I assume it is Dr. Grimes' opinion that the stomach can ordinarily be used to replace the esophagus in malignant disease—certainly when the malignant process is confined to the lower two-thirds of the esophagus. I think the management of replacement of the distal end of the esophagus in so-called peptic esophagitis is a somewhat different problem. When it is necessary to resect the distal end of the esophagus in peptic esophagitis, I believe that the proximal jejunum or transverse colon may actually prove to be a better replacement than the stomach when the results are reviewed after one or two years following the replacement procedure.

I thought it might be of interest to you to see what happens to a Roux–Y replacement in a patient who has survived nine years after total removal of the stomach and the lower end of the esophagus for carcinoma. This patient is one of the few lucky people who has survived a long period of time without evidence of recurrence of the neoplasm. Apparently we were able to remove all of this man's tumor in 1951 and the patient has remained very active for the past nine years. The esophagojejunal anastomosis is approximately at the level of the left inferior pulmonary vein.

A roentgenogram taken about seven months after the replacement operation showed the stomach totally removed along with the spleen and the distal end of the esophagus, and the proximal jejunum elevated in the Roux–Y fashion. By late in 1959 the jejunum which is above the diaphragm had become considerably dilated and was functioning more or less as a new stomach.

This patient occasionally has symptoms suggestive of mild peptic esophagitis. These symptoms are readily relieved by antiacids. It was necessary to place this patient on vitamin B_{12} because of an anemia approximately three years after the total gastrectomy. At the present time the patient is actively engaged in an occupation and supports his wife and four children.

Orville F. Grimes (closing): The technical points brought out by Dr. Brewer are extremely valuable and we should keep them most prominently in our minds at all times. Our experience with gastrostomy is not so unsatisfactory as his, and in some of our elderly patients with malignant obstruction of the esophagus, no other choice of therapy except gastrostomy followed by radiation was possible.

In these severely debilitated patients one can justifiably contest the advisability of a major substitution procedure to connect the mouth with the stomach for feeding purposes when an opening in the stomach serves to maintain nutrition and irradiation therapy may offer significant palliation.

I think we will all agree that in children, in whom the mesentery and vessels of the colon are long and sometimes even redundant, the use of the colon is successful and is the intestinal segment of choice for substitution purposes.

I suspect that the transposed intestinal segment, regardless of its type, will grow as the child grows. After all, the peristaltic activity of any intestinal transplant exists in its new habitat. This is in turn dependent upon its blood supply and as it grows in the abdomen, it should also grow in its new location.

Dr. Hunnicutt's technical contribution is an important one for us to consider. In using the ileocolon as a gastric substitute he anastomoses the esophagus to the ileum, rather than to the cecum, by choice. The ileum is the narrowest segment of the small bowel, and one wonders whether some degree of stenosis might be expected if the esophageal mucosa is turned upon itself before its anastomosis with the ileum.

Dr. Synder's comments are particularly apropos, because it is probably true that the stomach cannot be elevated into the chest sufficiently high to prevent esophagitis, if indeed such a maneuver does prevent esophagitis, unless the right thoracic approach is used.

I believe Dr. Stephens' comments are important. He states that when the esophagus must be replaced by segments of intestine of one sort and another it is probably wise to consider segments other than the stomach for the high esophageal strictures. I cannot entirely agree with this thesis.

The main point that I wish to emphasize is that we are frequently engulfed in a wave of enthusiasm for colonic, jejunal or some other type of replacement procedure, forgetting the value of the more easily mobilized and utilized stomach.

Tumors of the Hand

A TEN-YEAR SURVEY AND REPORT OF 437 CASES

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From the University of California Hospital and The Department of Surgery, Franklin Hospital, San Francisco, California.

TUMORS of the hand are many and varied and present interesting problems in diagnosis and management. Some tumors such as ganglia are frequently seen in the hand, while others such as sebaceous cysts are rarely seen. Certain tumors show a peculiar tendency to develop predominantly in the hand. Those include glomus tumors, implantation cysts, ganglia and the isolated xanthomas of tendon sheaths.

The etiology of most tumors of the hand is not clear. Heredity, disturbances in metabolism, and trauma have been cited as significant factors. It is generally agreed that surgical removal is the preferred treatment in the management of tumors of the hand and often necessitates skin graft replacement. Biopsies should always be of the excisional type. "Frozen section" studies immediately upon removal of the tissue specimen have proved invaluable, not only in diagnosis, but also in the determination of adequacy of margins to be removed with certain lesions.

This study reviews the experience of the senior author, the University of California Hospital, and the Franklin Hospital in San Francisco in the management of 437 tumors of the hand seen during the period 1949 to 1959. (Table 1.)

As anticipated, the vast majority of tumors of the hand encountered in this series was benign. Ganglia were the most common of the tumors, and accounted for one-third of the total number in our series. Malignancies of the hand occurred next most commonly, and comprised 24 per cent of the total. Inclusion cysts, giant cell tumors, and angiomas were commonly seen, and comprised another one-fifth of the tumors in this study. Tumors which occur less fre-

TABLE 1
TUMORS ENCOUNTERED AT THE UNIVERSITY OF CALIFORNIA HOSPITAL AND THE FRANKLIN HOSPITAL IN SAN FRANCISCO (1949–1959)

Type of Tumor	Number of Cases	Per cent of Total
Ganglia	146	33
Carcinomas	104	24
Inclusion cysts	37	
Giant cell tumors	32	22
Angiomas	26	
Fibromas	18	
Papillomas	10	
Mucus cysts	7	
Chondromas	7	
Lipomas	6	
Melanomas	5	
Acanthomas	5	
Glomus tumors	4	
Pyogenic granulomas	4	
Neurofibromas	4	
Plexiform neuromas	3	21
Fibrosarcomas	3	
Osteochondromas	3	
Traumatic aneurysms	2	
Synoviomas	2	
Myxofibromas	2	
Sebaceous cysts	1	
Leiomyomas	1	
Hamartomas	1	
Neurofibrosarcomas	1	
Giant-cell sarcomas	1	
Osteoid osteomas	1	
Metastatic tumors to bone (Hodgkins)	1	
Total	437	

quently made up the remaining one-fifth of tumors of the hand with fibromas, papillomas, mucus cysts, chondromas, lipomas, and melanomas occurring predominantly. In this presentation those tumors of particular interest will be classified and considered according to the tissues from which they appear to arise.

TUMORS ARISING FROM SKIN

Sebaceous Cysts. Sebaceous cysts of the hand [*21,29,31*] occurred only once in our series. These cysts are of interest on the hand because of their rarity and because they must be differentiated from other more common lesions of the hand such as inclusion cysts, ganglia, and other subcutaneous tumors over which the skin is freely movable.

Sebaceous cysts develop only on the dorsal aspects of the hands and are characteristically adherent to their site of origin on the skin. Complete excision should result in cure.

Epidermoid Cysts. Epidermoid cysts [*15,24*], or inclusion cysts, one of the more common tumors of the hand, occurred thirty-seven times in our series. In more than half the cases a history of trauma such as a puncture wound, laceration, crushing injury, or operative procedure was elicited with the cyst gradually appearing after a latent period of weeks, months, or years. The theory that epidermoid cysts may develop from the subcutaneous implantation of epithelial elements by trauma is now generally accepted.

Clinically, inclusion cysts usually produce no symptoms. They occur particularly on the palm and the volar aspects of the digits and often in amputation stumps, as firm, elastic, rounded tumors lying free in the subcutaneous tissues. The free mobility of the skin overlying the cyst and the lack of attachment to deep structures assist in differentiation of inclusion cysts from sebaceous cysts, ganglia, xanthomas, and other deeply attached tumors of the hand. Surgical removal offers an excellent prognosis for cure.

Mucus Cysts. Synovial or mucus cysts [*21, 29,31*] occur on the dorsum of the distal phalanges at or near the sides of the base of the nail as flat, broad, superficial cysts resembling ganglia. The vast majority is seen in adult females, and the middle finger is involved predominantly. Seven such tumors were encountered in our study. The fact that mucus cysts recur promptly after removal, unless the overlying skin and cyst are excised together, would indicate that these cysts originate from the lower layers of the corium.

Grossly, the skin covering the mucus cyst is often so thinned that the clear, gelatinous, ganglion-like contents of the cyst can be seen through this translucent epithelial covering. These mucus cysts usually cause no symptoms although their location over the base of the nail often leads to a grooved deformity of the nail. Successful treatment depends upon total removal of the cyst and the overlying skin, closing the defect with a small skin graft.

Carcinoma. Carcinoma is by far the most frequently occurring malignancy of the hand [*9–12,22,25,26,31,37,42,48,49*]. It occurred 104 times in our series and comprised 90 per cent of all malignancies of the hand. This lesion is usually seen on the backs of the hands in men in the older age group. The palm was involved in only two of our cases. The median age for squamous cell carcinoma of the hand in our patients was sixty-five years. The squamous cell variety occurred in the hand more frequently than the basal cell type in a ratio of approximately six to one.

A history of chronic irritation or trauma is elicited from 40 to 80 per cent of patients suffering from carcinoma of the hand. Exposure to sunlight and weather is probably the most frequent of these irritating factors although Mason's experience shows that exposure to small doses of irradiation over a prolonged period was the irritating factor in the majority of his cases.

The appearance of squamous cell carcinomas may vary from that of an unhealed fissure or crack to that of a well developed tumor. The lesion may be flat or indurated with ulceration, or elevated as a mass which resembles cauliflower. The tumor may exist undetected as carcinoma *in situ* as is frequently demonstrated in areas of x-ray dermatitis.

Most squamous cell carcinomas of the hand are superficial and of low-grade malignancy. However, those which ulcerate and invade deep structures grow more rapidly and have a greater tendency to metastasize.

The treatment of choice for carcinoma of the hand consists of early and wide resection of the local lesion with or without skin grafting, following which the patient should be observed closely for at least the ensuing two to three years. Large extensive lesions of the palm or dorsum may necessitate amputation of part or

all of the hand. Regional node dissection is indicated in all patients demonstrating enlarged lymph nodes as metastases may be expected in 50 per cent of such patients. In such instances it is our policy to perform axillary and/or epitrochlear node dissection approximately two weeks after excision of the primary lesion. Prophylactic dissection of regional nodes is not performed routinely by our group because of the relatively low incidence of metastases, and because of the older age of most patients suffering carcinoma of the hand. In our series only two of sixty-three patients demonstrated palpably enlarged axillary nodes with nodes being positive in one case.

Prognosis for carcinoma of the hand depends on the extent of the local lesion when first seen. Early lesions without metastases, treated adequately, offer an excellent chance for cure. If metastases are present the outlook is considerably less favorable. Our experience with carcinoma of the hand revealed a five-year survival rate of 48 per cent.

Melanoma. Melanomas of the hand [*2,13,16,20,21,29,31,35,40,41,52*] occurred five times among the patients in this study. One lesion involved the volar aspect of the left little finger, one the dorsum of the hand, and three were located in the subungual area (melanotic whitlow of Hutchinson), two of which were on the thumb. A definite history of trauma was present in two of the five cases.

Infection occurs early in subungual melanomas, and at the onset the true nature of melanoma is often not recognized, the lesion being mistakenly treated as chronic paronychia. The nail soon separates from the nailbed and cracks, allowing a fungoid, ulcerating tumor mass to appear which eventually destroys and replaces the nail. The tumor may arise from under the nail or from the sulcus at the edge of the nail. A black border or halo of increased pigmentation is usually present even in the amelanotic melanomas. Clinically, pain is not a prominent symptom. The patient complains only of the presence of the lesion, and, if ulceration exists, of an annoying brownish discharge which stains clothing.

Treatment of melanoma consists of early and radical excision of the local lesion including the deep fascia. In the case of subungual melanoma, the digit should be amputated [*31*].

Regional nodes were palpably enlarged and contained metastases in four of our patients with melanoma of the hand. Node dissection was performed in three of these patients and node biopsy was performed in the fourth who proved to be inoperable. The fifth patient demonstrated no nodes initially, but now has generalized metastases only eighteen months after biopsy was performed. This experience would indicate that prophylactic dissection of the axillary and epitrochlear nodes is indicated in the management of melanoma of the hand, and, according to Pack, may best be performed six weeks following excision of the local lesion. Forequarter amputations are extremely mutilating, and thus far have offered little more in regard to prognosis than have less extensive procedures.

From a prophylactic standpoint, it is important to point out that melanomas develop from junctional nevi in at least 65 per cent of cases. As pigmented nevi on the hand are almost always of the junctional type, their removal, particularly if on the palm or under the nail where they are exposed to trauma, seems indicated.

Although the prognosis for subungual melanoma is considered more favorable than for melanoma elsewhere, Pack reports a five-year survival rate with no evidence of recurrence for all subungual melanomas of only 18.5 per cent. Only two of our five patients are living and both have generalized metastases one and a half and four and a half years after diagnosis. Two patients died of metastases within four years of diagnosis. One patient with subungual melanoma died of coronary heart disease six years after diagnosis with no evidence of melanoma at autopsy.

TUMORS OF FIBROUS TISSUE ORIGIN

Ganglia. Ganglia [*6,20,21,23,31,37*], the most common tumors of the hand, occurred 146 times in our study. They occur more frequently in women and affect both hands about equally. A history of injury is obtained in approximately one-third of the cases. The etiology of ganglia is not clear.

Ganglia are seen most frequently on the dorsum of the wrist where they originate from the capsule of the intercarpal joints and the extensor sheaths. The second most common site of ganglia is the volar wrist in the region of the flexor carpi radialis tendon where ganglia develop from the underlying joint capsule and the fibrous sheath of this tendon. In this loca-

tion the radial artery is often intimately associated with the tumor and must be carefully protected during excision if damage to this vessel is to be avoided. Ganglia also occur quite typically in the distal palm and on the volar aspects of the proximal phalanges where they arise from the fibrous sheaths of flexor tendons. Ganglia are occasionally seen arising from the joint capsules of interphalangeal joints. Clinically, ganglia appear as single, multiple, unilocular or multilocular, encapsulated cysts containing a thick, clear, gelatinous fluid. They are usually quite readily recognized by their typical locations, and by the presence of a cystic swelling which is round, smooth, firm, and elastic. Ganglia are characteristically attached to their underlying site of origin on joint capsules, tendon sheaths, or ligaments. Symptoms usually depend on the size and location of the tumor. The patient may be disturbed only by the unattractive appearance of the tumor. Aching pain, weakness, and impaired use of the hand are not unusual complaints, and may actually precede the appearance of ganglia. Ganglia of the flexor tendon sheath appear as hard, tender nodules, and ordinarily interfere with the grasp.

The highest percentage of cures can be obtained only by complete surgical excision which gave a recurrence rate of 12 per cent in our series. However, the senior author has experienced recurrence of ganglia in only one case during the past ten years when the ganglion has been removed with a generous margin of tissue and with the ganglion left attached to its base. Aspiration of the cyst, followed by injection of Hydrocortone,® is not recommended in the management of ganglia as it led to recurrence in four of five patients so treated and resulted in severe septic arthritis of the wrist joint in one additional case.

Giant Cell Tumors. These are frequently seen on the hand [*17,20,21,29,31,33,43*] and occurred thirty-two times in our series. Their origin is not clear but they are probably synovial tumors arising from tendon sheaths or joint capsules. A history of trauma is elicited in about half the cases. Giant cell tumors occur most commonly on the flexor surfaces of the digits in middle aged individuals as asymptomatic, isolated, multinodular tumors which are firm, elastic, and non-tender. The skin is freely movable over these tumors, but they are firmly attached at their base. They are occasionally seen on the palm, the dorsum of the hands, and about the wrists. When exposed at surgery this multilobular lesion is easily recognized by its typical yellow, or yellow-brown color. The tumor is encapsulated. When it is in the finger it is usually densely adherent to the flexor tendon sheath. The tumor may also involve tendons, joints, and bone, and in one of our patients a giant cell tumor arose from the interphalangeal joint of a digit as a perfect mould of this joint space. Treatment of giant cell tumor consists of complete surgical removal including its attached base. The tumor, although benign, was found to recur in approximately 16 per cent of the cases in our study because of incomplete removal. None of the senior author's cases which were treated by radical surgical removal have recurred.

Fibroma. Fibroma of the hand [*21,29–31, 51*], which is considered rare, was encountered eighteen times in this series. The tumor may arise superficially from the skin, subcutaneous tissues, nailbeds, or deeply from such structures as tendon sheaths, joint capsules, ligaments, periosteum, and rarely from tendons. Clinically, fibromas usually develop insidiously as a nodular tumor which grows slowly. They are spherical or ovoid in shape and firm and elastic in consistency. They may be freely movable or fixed in position. Except for the presence of the tumor mass, they are ordinarily asymptomatic. Complete surgical removal should effect a cure. Recurrence of an apparently benign fibroma should suggest the possibility of sarcomatous degeneration.

Fibrosarcoma. Fibrosarcoma [*3,21,31,51*] occurred only three times in our series. One patient, a boy five years old, developed a fibrosarcoma of skin and subcutaneous tissue of the base of the right ring finger which has not recurred following wide local excision seven years ago. The second patient, a seven year old boy, developed a fibroma of a superficial type on the ulnar side of the hand which proved to be fibrosarcoma following its second recurrence. The tumor has not recurred following wide local excision ten years ago. The third patient, a twenty-eight year old man, developed a recurring fibroma of the distal phalanx of the right thumb in 1936. In 1952, following the fourth recurrence, notwithstanding local excision and progressive amputation of the thumb, a histologic diagnosis of fibrosarcoma was finally made. The entire thenar eminence was then

amputated and the patient is well without recurrence seven years later.

Clinically, fibrosarcoma may present as a deep swelling, but usually arises superficially as a hard, fixed, painful tumor, often reaching large size with a tendency to invade the skin and neighboring tissues. The tumor may grow slowly over a period of many years and then evidence rapid growth, or may be rapidly invasive from the start. Metastases to the lung by way of the blood stream and, less commonly, to regional nodes by way of the lymphatics, occurs early. Once fibrosarcoma is established, radical excision or amputation offer the best chance for cure. The tumors are resistant to radiation.

Lipomas. Lipomas of the hand [*5,21,31,46*] were seen in only six patients in this study. These lesions occur most frequently on the volar aspects of fingers or palms as soft, well encapsulated tumors. They usually appear superficially in the subcutaneous space but may be found lying within tendon sheaths occupying fascial spaces or infiltrating muscle. Diagnosis is suggested by the presence of a soft, pseudofluctuant, encapsulated tumor which has grown slowly without symptoms over a period of months or years. The application of an ice bag over the tumor may be an aid in diagnosis as the lipoma hardens as the fat becomes chilled. Treatment consists of surgical removal which, in the case of certain deep lipomas, may require an extensive and difficult dissection.

TUMORS OF NERVES

Only eight tumors in this study arose from peripheral nerves of the hand [*21,31,44*]. Four of these were neurofibromas, three were plexiform neuromas, and one was a neurofibrosarcoma.

Three of the neurofibromas occurred on the volar distal phalanges of digits while the fourth developed on the thenar eminence of the palm. All of these neurofibromas had developed on the flexor surface of the hand as smooth, encapsulated, asymptomatic tumors. Neurofibromas are only moderately movable under the skin and characteristically arise close to flexion creases and in close association with the cutaneous nerves.

In this study, one of the plexiform neuromas characterized by a diffuse thickening of the median nerve and its branches was encountered in a boy seven years of age. This patient demonstrated the nodular thickening of the skin and subcutaneous tissues of the palm and the hypertrophy of involved digits known as elephantiasis neromatosis. Two attempts at surgical removal performed elsewhere had been unsuccessful. In most cases, because of the extensiveness of the lesion, partial excision and skin grafting may be of benefit, particularly from a cosmetic standpoint. It should be emphasized that neurofibromas may develop deeply within a nerve trunk, giving the nerve a fusiform appearance. The median nerve is most frequently involved. In most instances the shiny encapsulated tumor can be easily shelled out from the nerve fibers spread out over its surface.

Neurofibrosarcoma was encountered in one of our patients as a small, hard flat, asymptomatic nodule located in the skin and subcutaneous tissue of the dorsum of the hand. Diagnosis was confirmed by histologic study. Wide excision and skin grafting were performed and now, four years after surgical removal of the lesion, the patient is living and well without evidence of recurrence.

Multiple neurofibromatosis of the hands associated with generalized cutaneous neurofibromatosis, as described by Von Recklinghausen, was not seen in this series. However, these lesions are important because of the fact that in approximately 15 per cent of cases, one or more of the tumors undergo sarcomatous degeneration. Early recurrence of nerve tumors of the hand strongly suggests sarcomatous degeneration. Certain neurofibrosarcomas may give the appearance of being benign clinically and histologically, and may grow slowly over a period of many years. Others are extremely malignant, metastasizing early and leading to death in twelve to eighteen months.

As with any sarcoma, the treatment is radical excision. The initial operation is most important and should be locally extensive. Amputation is often necessary.

TUMORS OF BLOOD VESSELS

Tumors arising from the vascular tree are not uncommon in the hand [*14,19,21,38,50*] and manifest themselves in a wide variety of forms ranging from simple telangiectases to the diffuse and progressive arteriovenous or racemose aneurysms. Twenty-eight such tumors of the hand were encountered in this series including twenty-five hemangiomas and three arterio-

venous fistulas, two of which resulted from trauma.

In the management of hemangiomas of the hand, the classification of Blackfield and associates has proved most practical, not only in classifying these lesions, but also in deciding upon the course of treatment. In this classification, hemangiomas are divided into two types, those that will undergo involution, and those that will not involute. The involuting or infantile type includes superficial hemangiomas (capillary and strawberry), the deep hemangiomas (cavernous), and the combined superficial and deep hemangiomas (capillary and cavernous). Hemangiomas of the involuting type are seen in infancy and early childhood and usually date back to the time of birth. These lesions grow progressively during the first nine to twelve months, reach a peak in development, and then begin to involute gradually. Complete involution can be anticipated in this group by the age of six years with treatment limited entirely to conservative measures.

The adult forms of hemangioma include port wine stains, true cavernous hemangiomas, venous racemose aneurysms, and arteriovenous fistulas which do not involute spontaneously and may require specific measures in therapy.

In the treatment of hemangiomas of the noninvoluting type, surgical removal is the preferred treatment. Radiation of sufficient degree to cause disappearance of the vascular elements will cause permanent tissue changes often necessitating excision and skin graft replacement. CO_2 "snow" also damages normal tissues and results in surface scarring. The injection of sclerosing solutions and tattooing are of limited value in the treatment of hemangiomas and are rarely used.

Cavernous hemangiomas showing growth with pregnancy or at puberty, and, if large and deforming, should be excised. However, cavernous hemangiomas are often so extensive that complete surgical removal is impossible. In such instances partial excision of the troublesome area in one or more stages may be of benefit utilizing skin grafts as necessary.

The treatment of venous racemose aneurysms, arteriovenous fistulas, and hemangiomatosis present formidable problems in surgery of the hand. Although few are cured, some benefit can be obtained by partial excision in one or more stages ligating as many of the vascular communications as possible. If the vessels of only a single digit are involved, amputation of that digit is the procedure of choice.

Traumatic Aneurysm. Traumatic aneurysms [*37,38*] were encountered on two occasions in this study. One resulted from laceration of the digital vessels of the thumb and the other from a crushing injury of the radial artery in the wrist and arm.

In such injuries, the hematoma which develops becomes surrounded with fibrous tissue to form a false aneurysm which manifests itself days or weeks after the original trauma as a soft, compressible, bluish, grape-like tumor which pulsates. Pain and tenderness are usually present if thrombosis has occurred. Excision of the aneurysm and ligation of communicating vessels resulted in cure in these two cases.

Glomus Tumor. Glomus tumor [*20,21,28,32,39*], although rare, is one of the most interesting tumors of the hand and occurred four times in this series.

This tumor develops from the neuromyoarterial glomus, a structure associated with the autonomic nervous system which assists in regulating body heat. About two-thirds of the reported cases have occurred on the upper extremity, and, of these, one-half are subungual. All four of our glomus tumors were located subungually. Their etiology is unknown although trauma was cited as a factor in two of our four cases.

Grossly, the tumor is well encapsulated and frequently measures 5 mm. or less in diameter, particularly if in the subungual location. Seldom does the tumor exceed 1 cm. in diameter. Multiple tumors have been reported. Clinically glomus tumors develop insiduously and grow slowly as small, painful nodules. The diagnosis should be suggested by the key symptom of pain which is usually severe and paroxysmal and which is usually triggered by pressure upon the tumor, or by changes in temperature causing sharp, lancinating pain to radiate up the extremity. The patient characteristically attempts to protect the part by cradling the hand in the opposite palm, or by keeping the hand in a pocket.

A careful search may reveal an area of reddish purple or cyanotic discoloration 3 to 5 mm. in diameter under the nail. The lunula of the nail may be blurred, and if the nail is inspected end on, it may be found to be slightly rounded in the transverse plane. Occasionally

erosion of the bony distal phalanx is seen on roentgengraphic study.

Associated vasomotor disturbances are common. A few patients have had a coincident Horner's syndrome and one of our patients demonstrated a severe syndrome like causalgia. Treatment consists of complete surgical removal of the glomus tumor following which relief of symptoms is dramatic.

Pyogenic Granuloma. Pyogenic granulomas [*21,29,31,34*] occurred four times in our series. They are considered by some to be true angiomas and by others to be simply heaped up masses of granulation tissue of infectious origin. A history of trauma or infection usually precedes the development of the lesion.

Clinically, pyogenic granuloma is seen on the volar aspects of fingers or palm, or about the nail bed as a mass of red, granulomatous appearing tissue with a stalk which presents through a small collar of epidermis. Exquisite pain and tenderness, and a ready tendency to bleed are characteristic features. Treatment consists of surgical excision of the tumor so as to include its vascular base, followed by suture of the small elliptical skin wound. The lesion is benign and does not tend to recur.

TUMORS OF BONE

With the possible exception of chondromas, tumors of the bones of the hand are quite rare [*1,17,21,27,29–31,33*]. In our series there were twelve such tumors including seven chondromas, three osteochondromas, one of which occurred under the nail, one osteoid osteoma, and one case of Hodgkin's disease which had metastasized to the bones of the hand. All but three of these tumors involved the phalanges and the remainder were located in the metacarpals. The lesions were found chiefly in the younger age group.

Enchondromas, bone cysts, and the rarely seen giant cell tumors of bone present a somewhat similar appearance clinically and radiologically. Treatment of all three lesions consists of surgical removal followed by thorough curettement of the remaining cavity. Packing of the cavity with bone chips may be necessary even though in some cases the space fills with clots followed by satisfactory regeneration of the bone shaft. Excised segments of the bone shaft should be replaced immediately with bone graft.

Osteoid Osteoma. Osteoid osteoma [*8,36*], first described by Jaffe in 1940, is an interesting entity, and occurred once in our study. The chief symptom is chronic, nagging, nocturnal pain which is characteristically relieved by small doses of aspirin. The distinguishing feature of osteoid osteoma on roentgenographic examination is the radiolucent central area surrounded by cortical thickening. Treatment of osteoid osteoma requires surgical removal of the tumor, taking care to curette out all of the central nidus of osteoid tissue to prevent recurrence. This tumor runs a natural course over a period of several years and eventually cures itself.

Exostoses, myxomas, fibromas, hemangiomas, and Ewing's tumor may occur in the bones of the hand. Bone sarcomas are extremely rare and the prognosis is poor. Treatment consists of amputation and, as in the treatment of all malignancies, should be proceeded by biopsy.

CONCLUSION

From this ten-year survey and review of 437 cases we have made the following observations:

1. Tumors of the hand are many and varied and present interesting problems in diagnosis and treatment.
2. Trauma may be a significant factor in their etiology and must be considered from an industrial injury, or medico-legal standpoint.
3. A knowledge of the frequency, location, and clinical characteristics of tumors of the hand is invaluable in diagnosis.
4. Benign lesions of the hand causing irritations may predispose to malignancy and should be removed.
5. Irradiation therapy is rarely necessary in the management of lesions of the hand and may be fraught with serious complications.
6. Tumors of the hand are best treated by surgical measures.

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DISCUSSION

JOHN KIRKPATRICK (San Francisco, Calif.): Due to the rarity of hamartoma of the hand, only a few cases have been reported. Therefore, I wish to illustrate that first. A growth on the hand of a baby boy had been noted within a month or two after birth, and he had been seen by several different physicians.

The baby is now six months old, and deformity has occurred. The middle finger and the ring finger are in contraction. The thumb is practically fixed in a position of non-function by a large mass which occupies almost the entire radial half of the hand. Roentgenograms show the distortion produced by the invasion of this tumor.

These tumors arise from primitive cells, including fat, connective tissue, smooth muscle and vascular structures. They are locally invasive and they do not metastasize. The progress of growth cannot be stopped and they are completely destructive.

Amputation proximal to the lesion is the only choice. Otherwise, it continues to grow into a large club-like tumor of the hand.

Dr. Butler also thought I should talk about some of the tumors and growths in the hand which are not related to the group of tumors that he has discussed, so I am going to discuss the granulomatous inflammations of the hand caused by the inanimate agents (chemical or physical) which may cause growths in the hand. These are oils, plastics, carbon, silica, rheumatic disease factor, collagen, cholesterol, and urea crystals.

It has been my experience that significant physical trauma does not produce a prolonged stimulative effect upon the cells of the reticuloendothelial system such as that which is produced by the agents just mentioned. The granulomatous inflammation, produced by such agents, singly or in combination, is prolonged. Its course may be only acute and reversible, subacute with remissions, or chronic and irreversible.

A sixty-four year old white man had recurrent painful swelling of the hands and feet for two years. On June 17, 1952, the hand became painful. No trauma was alleged. On June 24 the dorsum of the hand became swollen. Ten days later examination revealed a hot, red, very painful swelling over the dorsum of the third metacarpal phalangeal joint.

One fluctuant area; was aspirated. When the suction was applied a clear, pink fluid was obtained in the syringe. Upon breaking the suction effect, a little air got into the syringe or for some other unknown reason, this solution promptly crystallized into an almost solid media. Microscopically the solids were pure uric acid crystals.

This patient's blood serum uric acid was only 4.3 mg. per cent. Our laboratories tell you that that is normal, or within normal limits, but in this instance it wasn't.

This represents one of the mysteries of gout that has not been solved. A tremendous amount of work is being done on it. But how can a practically supersaturated solution of uric acid stay in the body in a liquid form, and immediately become crystalline upon extraction? It might help us with some of our problems with gout, if we knew the answer, and investigations are going on to try to find the reason for it.

A forty-two year old Negro awoke one morning with a painful swollen distal phalangeal joint. He thought he had a piece of steel in it. A roentgenogram taken three days later revealed an opacity near that joint. There was no evidence of any steel or other opaque foreign body. This was the solid phase of a uric acid granuloma in the region of the collateral ligament of the distal phalanx of this finger.

After prompt therapy with colchicine and salicylates the acute inflammatory phase subsided within about three days. Three weeks later an x-ray film showed that the deposit had been completely absorbed except for a slight shadow at the distal end of the collateral ligament.

A patient with subacute gout had persistent tophaceous nodules in the extensor tendon mechanism, along the dorsum of the hand and along the tendon expansion over the middle joints. This condition is characterized by acute attacks of inflammation with remissions and recurrences and additional deposits over many years, unless active therapy is given for gout. Strangely, these tophi select the ulnar border of the fingers more often than they do the radial side. We do not know why that happens.

The snapping middle joint that gives a sharp click when the finger is flexed usually has a tophus over the ulnar margin where the lateral band slips over the dorsum of the middle phalanx during flexion and extension. The deposit in the tendon produces a small protuberance, and when the tendon rides over the condyle of the proximal phalanx as it slides laterally, it snaps audibly.

A seventy-five year old man has chronic, irreversible tophaceous gout involving all the soft tissues and bones of the hand. It is now associated with kidney failure. He cannot excrete uric acid. He now has weeping tophaceous sinuses, and all of the structures of the hand are involved.

Surgical excision of urate deposits is indicated if the function of the hand can be improved by the surgical procedure. Constant medical treatment and supervision are necessary to prevent severe impairment of the function of the hand caused by gout.

JOSEPH H. BOYES (Los Angeles, Calif.): I would like to point out just two or three things which might be of interest. One is that there is an inherent

fallacy in collections of figures. This cannot be avoided. An extremely high percentage of Dr. Butler's patients had malignant lesions of the skin. Twenty-four per cent of them had carcinoma of the skin and six per cent had angiomas. If this same collection was made in one of the hospitals where I work, one would not find this high percentage of carcinoma of the skin, or the high percentage of angiomas.

Dr. Butler included in his 437 cases thirty-seven cases of inclusion cyst and four of pyogenic granuloma. This accounts for 10 per cent of the total. Should these be called tumors?

In his series he has only thirty-two cases of xanthoma, or giant cell tumor, which account for 7 per cent. This is extremely low compared to other series. I believe the one study from New York showed ganglia the most common tumor, and xanthoma the second most common.

My experience is particularly with the ganglia and the xanthoma. I have never found any evidence to prove that ganglia arise from a tendon sheath, from a joint capsule, or from the dorsal carpal ligament. The dorsal carpal ganglion, the common one on the back of the wrist, almost invariably arises from the ligament between the scaphoid and the semilunar bone. Unless this portion of the ligament from which the ganglion arises is removed, there will be a recurrence.

Ganglia also arise on the volar surface, and our figures show seventy-two cases on the dorsal and eighteen on the volar surface. On the volar surface, the radial side of the wrist is the most common site. These arise from the ligament between the distal end of the radius and the scaphoid. If one cannot trace the ganglion to that point, there may be a recurrence.

A few ganglia occur on the volar side of the wrist on the ulnar side, and do not present on the surface, but extend down beneath the volar carpal ligament and follow the course of the motor branch of the ulnar nerve. A large series of these has been reported in England. Seddon reported ten. We reported one about five years ago. We have treated two others since. We have also seen one volar ganglion involving the median nerve and causing a median compression. The important thing is that they do not present to the surface. Therefore, a patient who has a motor paralysis of the hand distal to the wrist without sensory changes may have this type of ganglion.

A patient had a paralysis of the motor branch of the ulnar nerve, without sensory changes. The motor branch had been displaced by the ganglion in this case.

This ganglion arises from the wrist joint, from the ligaments in the wrist joint, and if you do not follow it to its source there will be a recurrence.

Another patient had a ganglion which did not produce a motor paralysis but produced sensory changes in the ulnar nerve, on the little finger and the adjoining side of the ring finger.

The possibility of a ganglion at the wrist producing changes in the ulnar nerve or median nerve must be kept in mind.

It has been our experience that xanthomas, or the giant cell tumors, usually occur on the extensor surfaces of the digits. Dr. Butler's cases were mostly on the flexor surface. We believe that there is no evidence to show that they ever arise from a tendon sheath. These are tumors of joint capsules. Recurrence is usually due to superficial removal of the lesion. Some tumors arise from the distal joint; others have arisen from the middle or even proximal joints.

One should carefully evaluate lesions such as this which tend to show a high percentage of malignancy.

Edmond D. Butler (San Francisco, Calif.): I wish to thank the discussants for their additional remarks on this paper.

Dr. Kirkpatrick has spent a surgical lifetime in the study of metabolic diseases, particularly in the hand and specifically gout. In our area he has not only pioneered in this field, but I think he has also made most of us very conscious of this metabolic condition, not only in tumors but also in the traumatized hand that does not respond to usual therapeutic measures. I admonish anyone interested in this field to keep the possibility of gout in mind, and not to hesitate to get a determination of blood uric acid in such cases.

In the many series which we had an opportunity to review in preparing this paper the incidence of tumors varied from one classification to another but I think this variation is not too great and that ganglia are always the most common tumors of the hand. Except for the moderate variations, I believe one gets a proper perspective from the study of most large series.

I found Dr. Boyes' description of the ganglion in the volar ulnar palm most interesting. I have never seen one in this unusual location, and I will be on the alert for ganglia at this site.

Problems in Fluid Replacement and Cerebral Edema in the Management of Surgical Lesions of the Central Nervous System

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There are perhaps three fundamental differences in the solution to problems in water, electrolyte and protein balance between patients suffering cerebral diseases or undergoing operations on the brain and those with other afflictions.

Because of these differences the surgical management of lesions involving the central nervous system may be complicated by water intoxication or dehydration, cerebral edema of dangerous degree, serum hyperosmolarity independent of dehydration, salt depletion, and combinations of these which may produce syndromes readily confused with a primary intracranial complication.

Postoperative management of water, electrolyte and protein of the uncomplicated surgical case rarely becomes difficult if correct data are collected and well recognized principles followed. The patient with cerebral disease or injury, however, may best be served by observing these important differences.

Examples of disturbances in fluid balance which may have their foundation in part, at least, on one or more of the aforementioned differences may well be reviewed as they have been encountered on the neurosurgical service at the University of California Medical Center, Los Angeles.

THE BLOOD-BRAIN BARRIER

The first of these differences is accounted for by the physical-chemical-electrical phenomenon known as the blood-brain barrier, which probably has its anatomical substrate in the ground substance between the capillaries and the neuroglial feltwork [*14*]. This barrier differentially excludes from the brain fluid certain extraneous items which readily enter other body fluids; it is responsible for the marked increase in the time required for equilibrium to be reached between brain fluid and plasma in contrast to that required between other body fluids and plasma. This barrier explains why most methods applied *in vivo* to the measurement of changes in tissue water, both total and extracellular, are not applicable to the brain, since most tracer substances which are utilized for measurement do not cross the barrier fast enough or in sufficient volume to measure brain water accurately.

Brain edema may occur following the administration of fluid if the barrier has been broken by disease or injury, or if the solute given crosses the barrier readily and carries water with it, or if a hypotonic solution is administered, thereby permitting water to enter the brain.

Whereas the same relative amount of edema is well tolerated in other tissues, the danger of even small degrees of cerebral edema is recognized when it is realized that a 10 per cent encroachment upon the intracranial volume may so distort and compress its normal contents as to endanger survival. Ten per cent of intracranial volume represents a small volume which is of little significance when total body fluid is considered, but it is of utmost importance if limited to the cranial chamber. If the brain is injured, diseased in other ways, or operated upon, the barrier which protects it is broken and the tissue responds to insult with edema. Now, if we add a solution of 5 per cent dextrose in water to the vascular system, the dextrose, which under normal circumstances

crosses the blood-brain barrier slowly, will enter the damaged zone and carry water with it. Its rapid metabolism and eventual penetration of the barrier and cell membrane are followed by cellular hydration, and the edema of the brain is further aggravated. Sodium chloride, on the contrary, passes the intact barrier so slowly and enters the cell in such minimal amounts that even though it enters the diseased zone, it will lead to less brain hydration and less aggravation of brain edema than the dextrose-water solution.

The surgeon's objection to sodium chloride solution is based on the recognized tendency of the stressed patient to retain sodium and an obligate amount of water during and immediately following major surgical trauma. Whereas this has led to the sound practice of withholding salt during and after operation to preclude further water retention, it is particularly to be observed that the substitution of salt-free solutions of water at this time will aggravate cerebral edema and raise intracranial pressure. Therefore, it is to be avoided in neurosurgical work. In the case of cerebral injury, physiological salt solution, on the other hand, will do this to far less degree, and the same applies to sodium lactate solutions and certain other isotonic or, indeed, hypertonic solutions. The primary concern, however, is that water, as such, be withheld during and following operations for conditions in which brain swelling may be deleterious; and it should be withheld during the critical period of neurosurgical convalescence. In the absence of important renal disease, the balance is in favor of risking some edema in other parts of the body by giving physiological salt solution rather than risking the aggravation of cerebral swelling by administering solutions which cross the barrier and carry water inter- and intracellularly.

The reasons for the rise in intracranial pressure with the administration of salt-free water are not fully known. When glucose and water are administered, the rapid metabolism of glucose results in water being left in excess such as to act as a hypotonic solution thereby swelling both extra- and intracellular spaces [4]. Certain experimental evidence is at hand to suggest that with the administration of water alone the reason for the rise in pressure lies in an increase of the volume of cerebrospinal fluid and a slight change in the water content of the brain itself [17]. It will be remembered that the cerebrospinal fluid is looked upon as an extension of the extracellular or intercellular fluid spaces of the central nervous system.

A word of caution is in order, for just as we do not recommend solutions which aggravate brain swelling, neither is severe restriction of fluids desirable, since the signs and symptoms which may develop as a result may mimic a primary intracranial complication by the evidence of central nervous system depression which can progress to stupor or coma.

CASE 1. A sixty-three year old bartender entered the University of California Medical Center, on August 8, 1957, having had anosmia for one year, blurred vision in the left eye for six months, and convulsive seizures and headache, each of four months' duration. An episode of diverticulitis had occurred one year prior to admission, but otherwise his health had been good. Examination revealed an alert man, with left optic atrophy and a left homonymous hemianopsia. The anosmia was confirmed. Protein in the cerebrospinal fluid elevated to 108 mg. per cent and an abnormal spike and slow wave focus in the left anterior temporal area led to the eventual diagnosis of a meningioma of the olfactory groove for which he was operated upon in two stages.

The first stage was on August 14, 1957. The tumor extended from the cribriform plate back to the planum sphenoidum. Postoperative administration of fluid consisted of 5 per cent dextrose in ½ normal saline solution, Ringer's lactate and oral fluids. He remained awake and responsive throughout his convalescence. The second stage procedure took place three weeks after the first on September 5, 1957. In this seven-hour procedure all tumor was excised except that lying over the carotid arteries, optic chiasm and the infundibulum area. During operation, 700 ml. of 5 per cent dextrose in water and 200 ml. of 5 per cent dextrose in ½ normal saline solution were administered together with 2,000 ml. of whole blood which replaced that which was lost. On the morning of the first postoperative day the patient responded to instruction and opened his eyes to command. During the next eight hours the patient was given 1,225 ml. of 5 per cent dextrose in water intravenously. He excreted 350 ml. of urine per catheter in this same period. Throughout this period the patient became steadily less responsive by comparison with his early morning status such that by 3:15 P.M. he was unresponsive except to deep pain. Blood pressure had steadily risen, and his breathing had become more shallow and labored. His pupils were dilated to 5 mm. and unreactive. The performance of tracheostomy and ventricular tap and the administration of 30 gm. of urea were followed

by improvement, and by 8 P.M. the patient demonstrated a good grip with each hand upon command and responded to simple verbal instruction. Thereafter, the patient steadily improved and completed a normal convalescence.

Comment: In such a complicated case, the variables over which one has only incomplete control are numerous. The contrast between the postoperative course of each of the two stages of craniotomy was striking. The first almost acted as a control for the other. The deterioration of this patient to a dangerous level of depression coincided with the ill-advised administration of the dextrose-in-water solution. The process was a reversible one of edema as supported by the rapid response to corrective measures and the steady improvement thereafter.

The foregoing outlines the disadvantages of parenteral administration of water in a form which will aggravate brain swelling. Another example of how water may give difficulty in neurosurgical work is the syndrome of voluntary overhydration.

CASE II. A forty year old story analyst underwent the second of two operations for spasmodic torticollis on October 14, 1958, when an anterior cervical rhizotomy was performed from the C_1 to the C_4 levels. The operative procedure went smoothly. On the day of operation the patient's total twenty-four–hour fluid intake was 1,800 ml. and urinary output 600 ml. On the first postoperative day intake was 2,300 ml. and urinary output 1,050 ml. On the second postoperative day he was ambulatory, afebrile and diaphoresing slightly. The weather was exceedingly warm, and the patient, with encouragement forced the oral intake of liquids. By 3 A.M. on the third postoperative day the patient was confused, breathing in a labored fashion and had been fighting to get out of bed most of the night. Temperature was 37°C.; pulse, 80 per minute; respiration, 20 per minute; and blood pressure, 168/98 mm. Hg. He was alternately stuporous and agitated. There were no abnormal neurologic findings apart from the mental state. Assessment of fluid balance over the twenty-four hours immediately preceding his stupor and agitation revealed a urinary output of 750 ml. and an oral intake of 6,130 ml. Although profuse diaphoresis had occurred, the patient's packed cell volume was 43 per cent; serum chloride, 99 mEq./L.; sodium, 140 mEq./L.; and calcium, 3.6 mEq./L. Non-protein nitrogen was 25 mg. per cent and carbon dioxide 28 mEq./L. Fluid restriction was followed by rapid improvement and the patient was recovered by the fourth postoperative day. Although laboratory data did not confirm the existence of water intoxication, the excessive hydration was associated with the clinical picture of cerebral edema [*16*].

Comment: Should this patient have had intracranial disease, this added problem could have been far more serious in its consequences. The way in which the blood-brain barrier causes the brain to differ from other tissues in the passage of solutes and their accompanying water from the vascular system into the intercellular and intracellular spaces influences fluid therapy and cerebral edema.

IMPAIRED CONSCIOUSNESS AND ITS INTERFERENCE WITH EXPRESSION AND SATISFACTION OF FLUID NEEDS

Induced Hyperosmolarity

The second of these differences relates to interference with consciousness on the part of the patient as a result of his afflictions such that body need as expressed by thirst, for example, may not be strong enough to break through a clouded sensorium. The result is a lack of voluntary demand for needed fluid. Under such circumstances, the powers of self-preservation are weakened, and the patient becomes fully dependent upon the attending surgical staff to anticipate his fluid needs.

The inability of the somnolent or comatose patient to control his own fluid intake orally or make his thirst known would seem well accepted, but even an otherwise alert surgeon may himself be "stuporous" as to the significance of this circumstance and its ramifications. The following two case histories are illustrative.

CASE III. A fifty-one year old machinist suffered two attacks of subarachnoid hemorrhage which were found on appropriate study to be due to an anterior communicating arterial aneurysm. The patient was operated on and the aneurysm isolated, but the patient remained semi-comatose and hyperpyrexic until hypothermia was initiated. From the time of operation intravenous and intragastric urea was administered to control intracranial pressure. The urinary output was maintained at what was thought to be adequate levels. When seen in consultation on the fifth postoperative day, the following data were recorded. The patient was comatose with an induced temperature of 30°C. On the preceding day he had been given 2,900 ml. of fluid and had excreted 3,100 ml. of urine with a specific gravity of 1.007. Plasma

sodium was 170 mEq./L., potassium was 4.1 mEq./L., and blood urea nitrogen 153 mEq./L. (Exogenous urea was given.) On the fifth postoperative day an intake of 2,400 ml. and an urinary output of 1,450 ml. with a specific gravity of 1.012 were recorded. Serum electrolytes were as follows: sodium, 174 mEq.; potassium, 2.95 mEq. Blood urea nitrogen was 141 mEq.

Comment: The striking hypernatremia in this case suggests that although fluid intake seemed adequate, if gauged by urinary output, the latter was not a suitable guide. The reason was that in the face of the heavy solute load from the exogenous urea, the kidneys were obligated to excrete water (the usual diuretic effect of urea) but in amounts inadequate to clear all the solute load because of the inadequate fluid intake afforded. Had the patient been awake his thirst mechanisms would presumably have dictated a greater volume intake. The obligatory urinary water excretion led to dehydration. Although at postmortem this patient was found to have some tubular epithelial degeneration, it may be noted that even normal kidneys may be unable to handle large masses of solute presented to them in this fashion, and although a urine of maximum osmolarity is accomplished by the kidneys, hypernatremia supervenes.

Case IV. A fifty year old male sewing machine operator was admitted to the University of California Medical Center, with a two-month history of memory loss and disorientation. The patient was operated upon for malignant astrocytoma in the left temporoparietal area. Exogenous urea was administered at the time of craniotomy to combat cerebral swelling and was continued thereafter by vein or gastric tube. By the sixth postoperative day, a total fluid intake of 16,150 ml. was balanced against a urinary loss of 18,375 ml. with a net deficit of 1,725 ml. not including the insensible loss which over the six days may have totaled an additional deficit of 4,000 to 5,000 ml. At this time hematocrit, however, was 43 per cent. By the seventh postoperative day the patient had deteriorated with such an obtunded mental status as to suggest the presence of a postoperative hematoma. Exploration revealed no clot, and a relaxed dura mater was present. At this time a striking blood picture was revealed: white blood count, was 20,050 per cu. mm.; serum sodium, 197.2 mEq./L.; potassium, 4.36 mEq./L.; and blood urea nitrogen, 246 mEq./L. Once again reliance on the volume of urinary output as a guide to fluid needs was ill-advised. The kidney was obligated to excrete this amount of urine under the severe osmotic diuretic effect of the urea, and because of inadequate supplementary fluid abnormal amounts of solute were retained. There were, however, no external clinical signs of dehydration. The excessively high osmolarity of serum and extra cellular fluid undoubtedly contributed to, if not caused, the altered mental state seen on the seventh day, but at no time following the initial operation was the patient sufficiently awake to express what was probably present, viz., a thirst, which expression would have led to more adequate hydration.

Comment: Simple dehydration of a patient whose sensorium is clouded should also be avoidable just as surely as should repetition of the aforementioned experiences. It is a lesson to be learned that the addition of an extra nitrogeneous load, whether by the use of urea or by administering a high protein diet such as via tube feeding, may tax the kidney even to the point of working maximally despite the deceptive reassurance gained by noting the specific gravity of the urine or its volume. In the previous two examples of plasma hypernatremia and hyperchloremia, the normal potassium is noteworthy. Whereas this example of hypernatremia and serum hyperosmolarity may have been avoided by ample fluid replacement, yet in the literature there has been discussion of a primary form of hyperosmolarity which might be due to specific neural mechanism derangement.

In 1939 Allott [*1*] reported a series of cases of disease in various parts of the brain associated with abnormally high sodium and chloride concentration in the serum, low urinary chloride and sodium, and a high blood urea nitrogen plus some degree of dehydration. In 1948 Sweet et al. [*18*] presented four cases of disease of the parietal lobe with varying degrees of hyperchloremia and azotemia and, in one case, hypernatremia despite "good" urinary output. These authors raised the issue as to whether the location of the central nervous disease was related to the imbalance. Subsequent reports of similar type followed. The three cases reported by Cooper and MacCarty [*5*] demonstrated hypernatremia without clinical edema or dehydration and with urinary outputs of low specific gravity in "adequate" amounts. These authors reiterated the possibilities behind this complex of (1) acute diabetes insipidus, (2) manifestations of the "alarm reaction" in exaggerated

form, (3) abnormal nervous system control of the kidneys, and (4) the lesions of the central nervous system themselves. Higgins et al. [11] excluded the second possibility just noted by observing that in the general adaptation syndrome, hyperchloremia was associated with an increased urinary chloride concentration, whereas in their six cases of head injury and hypernatremia low urinary sodium and chloride was noted. They proposed a mechanism by which the chloride ion excretion via the kidney is reduced and noted the similarities to desoxycorticosterone acetate effects in man. Anthonisen et al. [3], in presenting two examples of the sodium and chloride retention syndrome following cerebral trauma, explained the disturbance, not on the basis of dehydration or renal dysfunction, but rather on central dysregulation. They proposed the following as possibilities: (a) a primary sodium chloride retention from increased mineral corticoids via the adrenal or pituitary, or (b) a primary water reduction resulting from disturbance of the osmoregulatory hypothalamic mechanism. As the latter irregularity occurred a decreased blood volume from inadequate water would result in active sodium chloride retention.

It is worth noting that in the case of Gurdjian and Webster [10], hyperkalemia accompanied the hyperchloremia and hypernatremia when, although fluids were forced, diuretics were also used, raising again the question of simple dehydration. The normal serum potassium content in our cases has been remarked.

It is suggestive in the review of many examples of hypernatremia in association with disease of the central nervous system cited in the literature that inadequate fluid intake may have contributed in large degree to the observed syndrome.

Let us take an example. If a patient weighing 70 kg. receives in his immediate postoperative state 10 gm. of sodium chloride, and because of increased intracranial pressure, 1.5 gm. of urea per kg. of body weight given twice in the twenty-four–hour period (a not unusual dose), the kidneys would receive 339 milliosmols from the salt and 3,570 milliosmols from the 210 gm. of urea, making a total of 3,909 milliosmols. The molecular weight of urea is 60; therefore 60 gm. of urea will equal 1,000 milliosmols or 17 milliosmols per gram of urea. If calculated on the basis of urea-nitrogen, 1 gm. urea-nitrogen equals 35 milliosmols (multiplying 17 milliosmols by the factor of $^{60}/_{28}$ or 2.14, since there are two atoms of nitrogen in urea, each with an atomic weight of 14). By calculating that 1 L. of fluid would be required for insensible water loss, the kidney would then require, even if maximally concentrating at 1,200 to 1,400 milliosmols per liter at all times, at least an additional 3,000 ml. of water to handle the urea and sodium chloride, a minimum total of 4,000 ml. of water. If, for any reason, the kidneys could not concentrate to this degree, then even more solute-free water would be required. These calculations do not account for the renal load of nitrogenous products of protein breakdown postoperatively.

The element of specific renal tubular damage accompanying the cerebral lesion or lesions was undoubtedly of major importance in the cases of Leutscher and Blackman [13]. Following the administration of sulfathiazole, their patients demonstrated hyperchloremia and hypernatremia during the stage of "recovery" from uremia. There was low urinary chloride and no acidosis. A similar case of sulfonamide nephrosis associated with encephalopathy was reported by Goodale and Kinney in 1949 [9]. Continued overhydration of their patient beyond the period necessary to relieve hyperchloremia led to death with a terminal hyperpyrexia of 42.5°C. suggesting unconfirmed cerebral edema.

In certain of these cases, and perhaps in ours as well, the excessive urinary output may have been aggravated by a mild deficiency of antidiuretic hormone, which in the face of an abnormally high serum solute load further aggravated dehydration by producing a polyuria [12].

The role of depressed consciousness on the protective thirst mechanism, as well as the role of excessive serum solute loads in patients suffering from disease of the central nervous system, has been nicely documented by Welt et al. [20].

INTERFERENCE WITH SPECIFIC NEUROHORMONAL MECHANISMS WHICH INFLUENCE FLUID BALANCE

A third main difference pertains to interference with specific neurohormonal circuits (known or probable) in the brain, the integrity of which is essential to the maintaining of body homeostasis. An explicit example is the supraopticohypophyseal antidiuretic hormone complex. Examples of less well confirmed circuits include the osmoreceptor system of Verney

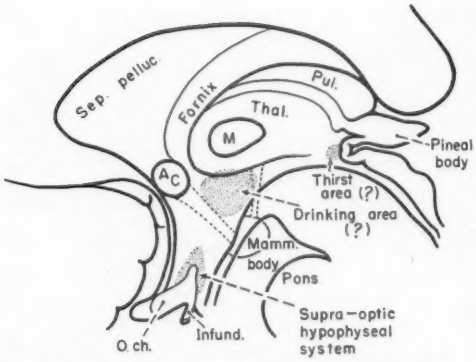

FIG. 1. Schematic composite presentation of established and possible hypothalamic and mid-brain areas concerned with water intake and output. Supra-optic hypophyseal system from Fisher, Ingraham and Ranson (Cat). Drinking area (?) from Andersson and McCann (Goat). Thirst area (?) from Gilbert (Rat).

[19], the drinking center of Andersson and McCann [2], a center governing thirst, and possibly others which might contribute to the salt-losing syndrome of Peters [15]. (Fig. 1.)

Interference with specific neurohormonal mechanisms by operative measures in the treatment of intracranial disease is most commonly attested to by the familiar syndrome of diabetes insipidus. Alertness to the possibility of its occurrence suggests the institution of well recognized methods of diagnosis and management. However, the combination of this syndrome with a disturbance of the central mechanisms for the control of thirst in an otherwise alert patient convalescing from major intracranial surgery can pose a challenge in fluid balance.

CASE V. A thirty-four year old dry-cleaning salesman, who had suffered with convulsive seizures for seven years, was diagnosed as having a large, left-sided parieto-occipital arteriovenous malformation. With the patient under hypothermia (29°C. minimum), the lesion was totally excised on March 4, 1959. The operative procedure required twelve hours, and vigorous bleeding occurred on several occasions. However, at no time was the patient's condition critical. During operation 3,750 ml. of whole blood and 1,400 ml. of 5 per cent dextrose in water were given. Except for transient arterial hypertension to 180/100 mm. Hg in the first hours of recovery the patient's immediate response was unremarkable.

Preoperatively the patient's blood analysis showed a hemoglobin of 14.2 gm. per cent; hematocrit, 44.7 per cent; white blood cell count, 16,175 per cu. mm.; and urinary specific gravity of 1.008. On the first operative day the hemogram was as follows: hemoglobin, 12.3 gm. per cent; hematocrit, 37.5 per cent; white blood cell count, 24,800 per cu. mm. By the third postoperative day the patient was taking all fluids by mouth, but from this day on oral intake fell behind output by 1,000 ml. The urinary specific gravity was 1.016. On the fifth postoperative day temperature was steady at 38°C., and the hematocrit 38 per cent. Oral fluids were encouraged, but no thirst was experienced by the patient. On the sixth postoperative day the patient's oral intake was 2,900 ml. and his output 3,800 ml. with a urinary specific gravity of 1.020. Until the tenth postoperative day, urinary output exceeded intake up to a 1,000 ml. differential. On the eleventh postoperative day Pitressin Tannate in Oil® was administered for the control of diuresis. The patient was advised to drink whatever he wished and in any amounts. He experienced no thirst but exhibited agitated behaviour at times. On the night of the twenty-third postoperative day, and seven days following simple cranioplasty, he tore off his night clothes, removed his head dressing, leaped over the side rails onto the floor, seized a pitcher of water and drank it. At this time mucous membranes were moist. Serum sodium was 138.7 mEq./L.; chloride, 103.9 mEq./L.; and potassium, 4.24 mEq./L. Hematocrit was 41 per cent. In the following four days voluntary intake rose to from 500 to 2,800 ml. above urinary output (Pitressin withheld), and on the last of these four days the patient drank 8,050 ml. and excreted 5,255 ml. in urine. On this day the patient again became aggressive and agitated, and generalized seizure occurred. Serum sodium was 125 mEq./L.; chloride, 91.9 mEq./L.; but the potassium was 5.13 mEq./L. Urinary specific gravity was 1.008 and the urinary sodium 20.4 mEq./L. A hypertonic salt test suggested less than optimal production of antidiuretic hormone, but, in addition, a disturbance in the thirst-regulating mechanism sufficient to permit the unbridled oral intake of fluid to the point of water intoxication was suspected. By rigid control of fluid intake and continued abstinence from exogenous Pitressin, the patient's fluid balance, electrolyte panel, and clinical status returned to normal.

Comment: The discrete lesion or lesions responsible for the complex of mild diabetes insipidus, plus a derangement of the thirst center, are unknown in this case. The severe amount of subarachnoid bleeding, remote effects of altering a large vascular mass, and manipulation of the brain during twelve hours

of intracranial surgery may well have produced reversible damage to such centers.

This patient had features not unlike the patient studied by Engstrom and Liebman [6] who, with diabetes insipidus, continued to take constant amounts of water orally and, unlike an uncomplicated case of diabetes insipidus, did not voluntarily reduce intake as urinary output fell. A deranged thirst-mechanism, together with diabetes insipidus, was in part responsible for chronic dehydration, hypernatremia and hyperchloremia.

Wolf [21] considers that osmoreceptors in the central nervous system similar to those postulated by Verney [19] lie on the afferent side of a thirst reflex such that an increased effective osmotic pressure of body fluids or a decreased cellular content of water or both, when present in sufficient degree, cause thirst. In man the upper limit of thirst threshold was calculated by Wolf to be a 1.23 ± 0.48 per cent decrement of cell water content.

In relation to the apparent lack of thirst in the patients receiving high doses of exogenous urea, Gilman [8] found that in dogs identical increases in osmotic pressure produced by sodium chloride caused apparent thirst and water intake, whereas urea produced no apparent thirst or fluid intake.

Although drinking and thirst may be closely allied, it is possible that the two may be dissociated, a possible explanation for some of the disturbances of intake seen in our patients. Andersson and McCann [2] found that in goats they could produce thirst and overhydration by microinjections of 3 per cent sodium chloride into the hypothalamus or by electrical stimulation between the descending columns of the fornix and the bundle of Vicq d'Azyr. Gilbert [7] produced in rats a marked reduction in voluntary water intake to the point of death by producing destruction of the subcommissural organ in the upper mid-brain.

A salt-losing syndrome in association with cerebral disease was described by Peters et al. [15]. Welt et al. [20], in presenting two such cases, suggested that the primary defect in this derangement is not in the anterior pituitary-adrenal cortex responses but an inability on the part of the proximal tubule to reabsorb salt properly, which inability presents a large salt load to the distant tubule producing a constant osmotic diuresis. The only examples of this condition in our experience were found in one case of total surgical hypophysectomy and another in a patient suffering from a tumor in the vicinity of the pineal body.

Case v. A ten year old schoolboy was admitted to the University of California Medical Center, on September 14, 1959. Except for a Bell's facial palsy in 1956 he was well until the onset in November 1958 of polydipsia of 2 gallons of fluid per day with a corresponding output. In June 1959 he became listless; by August he was noted to have an intermittently stiff neck. In the two to four weeks prior to admission, double vision and episodes of black spots before his eyes were noted. Upon examination he was a bright boy with a high grade of bilateral papilloedema and enlarged blind spots. His pupils were large and reacted sluggishly to light on distant gaze. A mild defect in eye elevation was noted. The diagnosis of an associated diabetes insipidus was confirmed.

On September 22, 1959, operation disclosed a tumor mass arising in the pineal region, indenting the posterior third ventricle, and producing a moderately severe obstructive hydrocephalus. Ventriculocisternostomy was performed and a full course of radiation administered. During twenty-four hours following operation the patient's urinary output ranged from 300 to 700 ml. per hour; and by 5 P.M. on the day of surgery he was hypernatremic with a serum sodium of 156 mEq./L. and a low urinary sodium of 15 to 20 mEq./L. Adequate hydration with electrolyte-free fluid and the administration of aqueous Pitressin succeeded in raising the urinary specific gravity from 1.001 to 1.017 and reducing its volume to 200 ml. per hour. The patient was receiving cortisone acetate twenty-four hours preoperatively, and postoperatively received doses of 25 mg. every twenty-four hours. During the first postoperative day fluids of hypotonic composition were continued to match the urinary output and provide for insensible loss. His twenty-four–hour intake was 4,155 ml. and measurable output was 3,600 ml.

On the morning of the second postoperative day the boy suddenly became shocklike, blood pressure was unobtainable, and pulse was 40 per minute. He responded to treatment but was more depressed than previously. Serum sodium at this time was 123 mEq./L. Prompt intravenous administration of soluble cortisone, blood and oxygen was followed by a prompt response of blood pressure. Parenteral sodium and potassium were given. Thereafter, sodium content of the urine decreased despite an associated low serum sodium. For example, on the fourth postoperative day in a twelve–hour period serum sodium fell from 136 to 127 mEq./L. during which time sodium intake was 357 mEq. and urinary output 384 mEq. (211

mEq./L.). By 7 P.M., serum sodium had fallen to 121 mEq./L. in the face of 50 mg. of cortisone administered per day. Desoxycorticosterone acetate was substituted, but positive sodium balance was attained only with an increased intake of sodium to match or exceed output. The fluids which were administered contained 220 mEq. of sodium per liter. With the further substitution of 9 alpha-fluoro-hydrocortisone and prednisone for other steroid therapy, and the maintenance of an intake of 233 mEq. of sodium and 106 mEq. of potassium per day, the patient stabilized, steadily improved, and was finally discharged on November 19, 1959, without any supplementation or other therapy required. It was evident from his December 1959 follow-up visits that his condition was stable, reflecting the reversibility of the striking salt-losing mechanism.

Comment: It is possible to suggest that the defect in this boy was due to a specific neural circuit derangement, but its location, if it does exist, is obscure. The possible remote effects of the severe hydrocephalus detract from placing too much attention upon the actual site of the tumor. In fact, the diabetes insipidus itself was possibly due to an indirect effect.

SUMMARY AND CONCLUSIONS

There are several important principles and differences to be observed in the fluid and electrolyte therapy of patients undergoing neurosurgical procedures on the central nervous system as compared with other surgical problems.

1. Because of the phenomenon of the blood-brain barrier and the potential danger of small amounts of excessive brain water in the production of edema the following cautions should be noted. (a) Hypotonic intravenous solutions should be avoided. (b) Solutions such as dextrose in water, the solute of which is rapidly metabolized, should be avoided since a resultant hypotonic or solute-free water will aggravate cerebral swelling. (c) Solutions the solutes of which readily cross the blood-brain barrier should be avoided.

2. The clouding of consciousness by the primary cerebral disease, or the operation for it, removes the ability of the normal drinking pattern and thirst mechanism to express themselves by protective voluntary intake. Reliance upon the urinary output may in itself produce a false sense of security. The solute load which the kidneys are required to excrete will produce an obligatory water loss and, at times, a low specific gravity, as well as produce or aggravate a state of acute dehydration. Most examples of postoperative hyperosmolarity have some degree of dehydration contributing to the cause. The administration of cerebral edema-combatting agents, such as urea, requires special attention to fluid replacement (of the non-barrier-crossing kind) to prevent dehydration.

3. Interference with specific neurohormonal mechanisms may lead to severe complications in fluid balance. The combination of a deficiency in antidiuretic hormone (A.D.H.) plus a deficient thirst center or drinking mechanism may, even in an alert patient, lead to severe water depletion. Water intoxication may occur with excessive, uncontrolled voluntary water ingestion due to disturbances of such centers. Severe electrolyte depletion, particularly if associated with diabetes insipidus, may follow the occurrence of a sodium-wasting syndrome in association with cerebral disease.

4. A careful charting of total intake and output is recommended. Hourly urinary output and specific gravity or osmolarity measurement will be helpful if antidiuretic hormonal deficiency is suspected. Calculation of the osmolar load given to the kidneys from the administration of protein or urea will indicate the amount of added water needed beyond basic requirements. Urinary output will not be a reliable guide to the adequacy of hydration. Measurement of serum electrolytes, particularly sodium chloride and blood urea nitrogen, will anticipate the occurrence of hyperosmolarity. The presence of renal disease or an altered pituitary-adrenal function should be ascertained preoperatively.

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DISCUSSION

JOHN RAAF (Portland, Ore.): We have been very much interested in urea the last couple of years and this material has proved to be of great value in the reduction of increased intracranial pressure. Many times the surgeon encounters a tight brain and he is unable to expose the necessary area because of swelling of the brain.

We have been using urea about two years and it has been on the market approximately one year. When we began its use, we worried about the electrolytes that might be excreted during the marked diuresis and because we could find little in the literature to help us out on this problem our resident in neurosurgery, Dr. Michael Mason, undertook this study. He has summarized the results of the diuresis of ninety surgical and non-surgical patients during the last two years.

Even though diuresis of 1,500 to 4,000 cc. of fluid occurred during twenty-four hours, serum sodium, serum chloride, alkali reserve and potassium stayed within approximately normal limits in the average person. Occasionally in a patient who was severely dehydrated there would be some alteration of the serum electrolytes.

The sodium concentration in the urine was summarized in four groups of patients. One group of patients represented the controls. They were non-surgical patients and were used to test the effect of diuresis without the superimposed stress of trauma or surgery.

The second group represented neurosurgical patients undergoing craniotomy, primarily, for brain tumor.

The third group was comprised of patients undergoing craniotomy for intracranial aneurysms and these patients were operated upon under hypothermia.

The fourth group consisted of patients who received steroids pre- and postoperatively, most of whom underwent hypophysectomies for breast carcinoma.

In the control series, there was a prompt decrease in the urine sodium concentration with a return to normal about the second postoperative day. Those patients who did not have steroids and did not undergo surgery under hypothermia, in other words the general run of neurosurgical patients for brain tumor, had a marked retention of sodium with diuresis for approximately four postoperative days. This retention of sodium is consistent, of course, with the normal stress response to a surgical procedure.

In patients who underwent hypophysectomy and were receiving steroids, there was even more marked retention of sodium. Patients undergoing surgery under hypothermia also showed retention of sodium.

The urine potassium concentration presented a different type of picture. In all four groups of patients there was a marked decrease in the urine potassium concentration and quite a prompt return within the first twenty-four hours.

Our conclusion, therefore, was that in patients receiving urea the best intravenous replacement fluid was dextrose in water, with perhaps 20 to 40 mEq. of potassium added.

We know that patients with uremia sometimes have an increased tendency to bleed and we were worried about that aspect when we first began to administer urea. We checked the Lee-White coagulation times, clot retraction time and prothrombin time in these ninety patients. There was no significant alteration noted in the Lee-White coagulatime time, and the clot retraction time was approximately normal. However, following diuresis in 60 per cent of our patients prothrombin time was abnormal. We are investigating this phase of the problem at present.

The blood and plasma volumes were measured and in many patients there was a decrease of as much as 1,100 cc. in the blood volume.

We believe that urea is a valuable adjunct and think dextrose in water is the fluid to give after dehydration with urea.

Roy Cohn (Palo Alto, Calif.): Dr. Stern's paper serves to call our attention to the complexities of fluid balance in patients with neurosurgical problems. During the last twenty years, as knowledge of electrolytes and body fluids has increased rapidly, numerous formulas have been worked out to simplify the handling of patients who had a disturbance of fluid balance. From Peters book in 1935 to Tovey's monograph in 1957, the rules have changed as technical methods have improved and different basic figures have appeared as, for example, the percentage of total body water.

Complicated as the problem is in neurosurgery, the problem is further complicated in the patient with head injury who also has a fractured extremity and a possible abdominal injury.

Unfortunately the oxygen requirements of the brain increase in shock, according to Kovach. The hexokinase activity in the brain increases as well. In muscles on the other hand glucose and enzyme activity slows down. Solutions which aggravate cerebral swelling such as dextrose in water are much less likely to cause pulmonary edema than saline.

The direct effects of cerebral trauma on mucous membrane permeability are yet another poorly understood factor in the problem of fluid balance.

Once again the clinician surrounded by a mass of knowledge both overwhelming in quantity yet insufficient in complete understanding, must rely on his clinical judgment to sort out properly the laboratory data. In general, I believe, more errors are made of overtreatment in this particular problem than undertreatment.

W. Eugene Stern (closing): Certainly Dr. Cohn put the finger on the sensitive spot when it comes to assessing the priority of treatment. My only plea would be that in the case of the confined spaces of the intracranial chamber, the brain will tolerate but a small amount of swelling, whereas the soft tissues in the other parts of the body may tolerate more swelling, and I would urge that the brain be taken into consideration when the patient "injured as a whole," as we are told these days, is cared for, whether it be by a general surgeon or the neurologic surgeon.

Certainly the administration of urea has been most helpful in combatting edema. One can calculate what the total requirements are in terms of fluid if one figures that there are about 17 mOsm. per gram of urea. If you administer the dose of urea at about 1 to 1½ gm. per kilo of body weight you can readily figure out what the milliosmol load will be to the kidneys, and if the kidneys function to capacity and excrete about 1,400 mOsm. per liter, one can calculate how much additional fluid is necessary incident to urea administration thereby preventing the consequences of dehydration.

There is a balance, then, between the acute osmotic effect of the urea in shrinking the brain on the one hand, and the replacing of the fluid which is necessary to prevent general body dehydration on the other. I think the urea would win; the brain would shrink. One could still use 5 per cent dextrose in water, probably, for this fluid replacement. Other than in the face of administration of urea, I would urge that the danger of brain swelling be given further priority, and that 5 per cent dextrose in water be withheld and replaced by either a physiologic salt solution or perhaps the compromise solution of 5 per cent dextrose in ½ normal saline solution.

Degenerative Arthritis of Lumbar Intervertebral Joints

A CLINICAL STUDY

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THE vertebral column is an extremely complex structure. Each lumbar vertebra has four distinct synovial joints related to its movement as well as intervertebral discs above and below. Each thoracic vertebra has ten synovial joints concerned in its movement in addition to the disc above and below. In all, the mobility of the bony spine depends upon 134 separate and distinct joints from occiput to sacrum.

This anatomic complexity, the lack of dissemination of knowledge concerning the pathology of the spine, and general disinterest on the part of physicians have led to many misconceptions concerning the cause and treatment of intrinsic lumbar spine disease. Even today patients and some doctors speak of lumbago and sacroiliac displacement as if such entities actually existed.

The medical literature is filled with articles about so-called disc surgery, but sparse attention has been given to clarification of the basic disease which is degenerative arthritis in lumbar intervertebral joints. Some significant contributions have been made.

Puschel [*1*] in 1930 pointed out that the intervertebral disc reaches its maximum state of development by the end of the twentieth year of life. Thereafter, the water content of the disc diminishes leading to lessened compressibility and to gradually increasing degenerative change.

In 1934 Mixter and Barr [*2*] published a classic article stressing the importance of posterior herniations of lumbar discs as an important cause of sciatic pain. The enthusiasm with which this concept was accepted has led unfortunately to an insistence by some that all sciatic pain is due to this cause.

Lumbar intervertebral articulations are compound joints. The disc itself is an amphiarthrosis, a joint with a ballbearing-like action. The posterior zygoapophyseal articulations are true joints. Their bony surfaces are covered with hyaline cartilage and the joints are lined with synovia and bound together by a fibrous capsule. The careful anatomic dissections of McRae [*3*] and Harris and MacNab [*4*] have clarified the degenerative changes which occur in the posterior joints. Degeneration of articular cartilage, osteochondral fracture, subluxation of joints, synovial and capsular thickening, loose body formation, periarticular lipping and adhesion formation have been noted. Indeed, these are the pathologic changes which characterize osteoarthritis in true synovial joints anywhere in the body.

Although the lumbar disc itself can be a source of pain as shown by Hirsch [*5*] in 1948 and Lindblom [*6*] in 1950, degeneration of the disc produces clinical symptoms usually by associated involvement of facet joints, spinal ligaments and nerve roots.

The present study is not intended to be a description of degenerative disease in the lumbar spine of the aged. Unquestionably degenerative changes in the spine are a normal part of the aging process. However, this process is not predictable as to time or decade of life. Premature or accelerated degeneration of one or more joints, or hereditary predisposition to early degeneration of one or more joints is unquestionably pathologic.

METHOD OF STUDY

Two hundred and ninety-eight patients were chosen for this study. Each was examined by me during a four-year period from 1955 to 1959.

FIG. 1. Advanced degeneration of the lumbosacral intervertebral disc in a thirty year old woman.

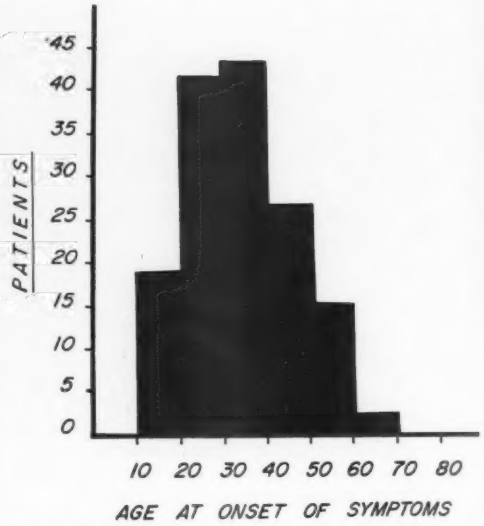


FIG. 2. Graph showing the age of patients at the onset of symptoms.

However, some have been followed up by me or my associates for as long as twenty years. There were 164 females and 134 males. One hundred twenty-five of these patients, all with lumbosacral arthritis, have been reported on in an earlier study [7].

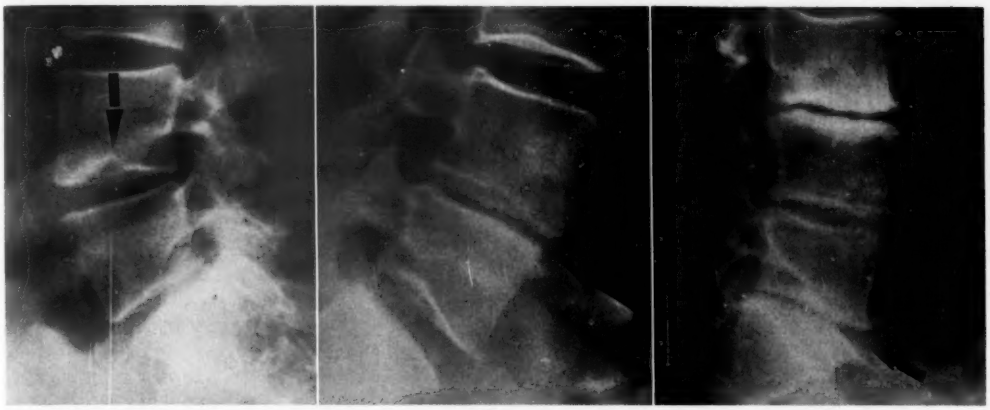
The lumbosacral joint, subject to the greatest amount of superincumbent body weight, is the commonest site of involvement. (Fig. 1.) Two hundred ten patients in this series had involvement of the lumbosacral articulations alone. Sixty patients had involvement of the fourth and fifth lumbar intervertebral joints. Sixteen patients had involvement of the fourth lumbar intervertebral joints only. Six patients had involvement of the third lumbar intervertebral joints only and six patients had involvement of the third, fourth and fifth lumbar intervertebral joints.

An hereditary predisposition to degeneration of the lumbar joints has been documented in sixteen families in whom it has been possible to examine siblings or relatives of successive generations. Thirty-three patients were treated for injuries in industrial accidents. Roentgenograms taken at the time of accident showed pre-existing degenerative joint disease in the lumbar spine in twenty-eight of these patients. The disease was confined to the lumbosacral or fourth lumbar intervertebral joints or both.

These patients and, whenever possible, their families were questioned repeatedly so that histories of a high degree of accuracy could be obtained.

Of the 298 patients, 148 were aware of their age at the onset of symptoms. Of the 148, 106 noted pain or other discomfort before the age of forty years. Eighteen of these patients noted symptoms between the ages of ten and twenty years. (Fig. 2.)

The nature of onset of symptoms is of interest. One hundred seventy-nine patients were able to describe the first symptoms of their disease. Thirty-nine complained of a dull low backache without significant radiation. Sixty-two patients suffered an acute attack of low back pain with or without sciatica from a simple body motion usually bending over to pick up some light object from the floor or to open a bureau drawer. Persistent symptoms developed in nineteen patients after falls either down stairs, from a height, or when thrown from a horse. All landed on their buttocks with force transmitted to the lower lumbar spine. Each patient was incapacitated after the initial injury for one to four weeks because of severe low back pain and muscular spasm. Fifty-four patients had joint injuries from significant physical strain either in contact sports, after childbirth, or by lifting



3A 3B 3C

FIG. 3. A, degeneration of the fourth and fifth intervertebral joints in a fifty year old woman with a ten-year history of back pain. The arrow points to an area of intracorporal disc herniation. B, degeneration of the fourth lumbar disc with anterior vertebral spurring in a forty-six year old woman. C, advanced degeneration of the third, fourth and fifth lumbar intervertebral joints in a sixty-two year old man with a thirty-year history of back pain.

weights in excess of 100 pounds. Five patients injured the lower part of the spine in automobile collisions. The duration of symptoms prior to the first examination varied from one week to forty years. The patients had been treated in many ways by physicians of varying disciplines. Supports, drugs, exercises, heat, traction, manipulation and surgery had been employed. Careful review of the patients' histories indicates clearly that the type of conservative treatment makes little difference in the early phase of degeneration of the lumbar joints. Acute symptoms subside in a week or two regardless of what is done unless a frank herniation or extrusion of the disc exists. Manipulation in the early stages of the disease often gave temporary relief but as the degenerative process progresses such treatment either gave no relief or aggravated symptoms.

Of those patients venturing a diagnosis at their first examination, almost all with a lengthy history stated voluntarily that their sacroiliac joint slipped in and out of place. This misconception of sacroiliac displacement as the cause of low back pain remains firmly entrenched in the minds of many patients and physicians alike.

The type, duration and frequency of acute attacks of pain have been noted. Acute and chronic symptoms have been differentiated. Whenever possible the progress of the disease has been recorded by serial roentgenographic study. Treatment, whether conservative or surgical, has followed certain definite concepts so that an opinion of some value can be given concerning response of patients to care rendered.

Careful scrutiny of the patients in this study reveals promptly that the physician cannot localize accurately the level of joint degeneration by history or physical examination alone. Back symptoms are similar regardless of whether the third, fourth, or fifth lumbar intervertebral joints are involved. (Figs. 3A and C.) If branches of the lumbosacral plexus are irritated, it is often possible to localize the level by physical signs alone.

Whether acute back pain or chronic backache characterizes the onset of the disease, its progress is marked by recurring attacks of pain at intervals of several weeks to several months. While the actual attack usually occurs from a simple body motion, it is often preceded by chronic fatigue, respiratory infection, a period of confinement to bed for an unassociated illness, by diminution in general muscular strength, or by emotional strain.

In the early stages of the disease the patient is often entirely free of symptoms between attacks. However, as the intervertebral disc narrows, the posterior zygoapophyseal joints undergo degenerative changes and chronic symptoms appear. By far the commonest of these is stiffness in the lower back upon arising

from bed in the morning. Some patients awaken with low backache to such a degree that they must arise and move about to gain relief. Others are comfortable lying flat in bed, but the act of arising from the recumbent position is painful, difficult and time-consuming. Upon reaching the upright position, the patient has great difficulty bending forward to wash his hands and even greater difficulty bending over to tie his shoes. Morning stiffness persists for thirty minutes to two hours and then subsides gradually.

Other significant chronic symptoms are backache with prolonged sitting or standing, lower back pain upon arising from sitting, pain while driving a car and pain in the lower back with light physical exercise.

Emphasis is placed upon the chronic symptoms of degenerative arthritis of lumbar intervertebral joints, for these symptoms inflict hardship upon the daily life of a patient. They must not be underestimated by the physician. A salesman with degenerative lumbar joint disease has great difficulty driving long distances. A housewife finds she cannot stand at a sink to wash dishes because of pain in the lower back. She has even greater difficulty while making a bed. A young mother experiences low back pain while lifting her infant from its crib.

Some patients have almost constant low backache which is punctuated by acute episodes of back and leg pain.

Inman and Saunders [8] in 1944 pointed out that localization of pain from deeply placed skeletal structures is poor. Furthermore, the response of deep structures to painful stimuli is variable. Hence, pain from degenerated lumbar joints may be felt in the lumbosacral area, over either or both sacroiliac joints, over the coccyx, in the inguinal area, in either buttock, in the lower abdominal quadrants, or if the fourth or fifth lumbar or first sacral nerve roots are irritated true segmental pain may be felt in the lower extremities. Rarely is leg pain the only symptom of this disease. Unusual radiation of pain from the lower three lumbar intervertebral joints has led to the unnecessary removal of pelvic organs in the female and to coccygectomy. One patient, a forty-five year old man, was subjected to cholecystectomy, reconstruction of the common duct, rectal surgery, bilateral varicose vein stripping, and bilateral epididymectomy in attempts to relieve pain due to a degenerated lumbosacral joint.

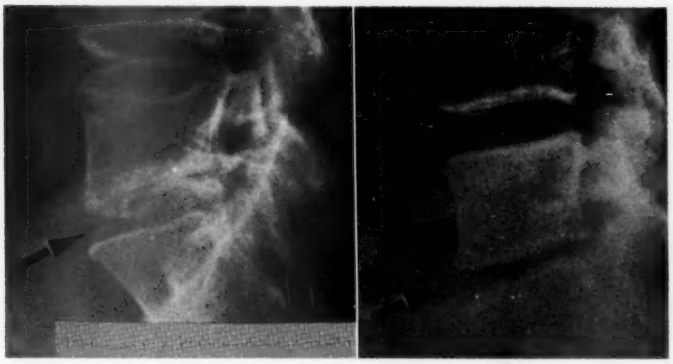
Patients examined during an acute attack manifest marked stiffness in the lumbar spine due to paravertebral muscular spasm. Restricted raising of the straight leg may be noted whether or not true sciatic nerve involvement exists. Irritation of the fourth or fifth lumbar or first sacral nerve roots causes reflex change, sensory disturbances and occasional muscular weakness in the extremity. There seems to be no direct relationship between collapse of the disc space and leg pain. Some patients with complete collapse of one or more disc spaces have never had leg pain. Others with a lesser degree of narrowing of an intervertebral disc have had severe sciatic pain. Also, the signs of irritation of the nerve root vary. They may be present in one attack and absent in the next.

Patients examined in the chronic stages of their disease demonstrate remarkably few physical signs. Slight stiffness in the lumbar spine, some difficulty arising from a chair or the examining table, local tenderness to deep palpation over the spinous process of the involved vertebra or a depressed or absent Achilles reflex may be the only findings of any significance.

Adequate roentgenograms are absolutely essential in making an accurate diagnosis as to the level and extent of disease. Positive roentgenographic findings may lag behind the physical signs by several months or even years. Serial studies are often needed to determine the exact type of joint degeneration.

The routine anteroposterior and lateral recumbent views of the lumbar spine are not enough. These must be supplemented by standing lateral views taken in flexion and extension. Oblique roentgenograms are necessary to determine the extent of degeneration in the posterior zygoapophyseal articulations. Carefully coned lateral views of the lower three lumbar interspaces are also needed. These views are difficult to obtain, and the technician must learn to position the patient with great care. (Fig. 4.)

The earliest roentgenographic sign of degeneration of the lumbar joint is slight narrowing of the intervertebral disc space accompanied sometimes by minimal spur formation at the anterior vertebral margin. Over a period of several years, the disc space gradually narrows



4A 4B

FIG. 4. Two roentgenograms of the lumbosacral joint of the same patient which show the importance of well centered films in making an accurate diagnosis. A, the incorrect position is shown. B, the correct position is shown.

and sometimes collapses completely. The facet joints undergo progressive degeneration. Subluxation of these joints can be demonstrated on well centered roentgenograms as can the degeneration of articular cartilage, eburnation of the subchondral plate, and periarticular lipping of bone. Occasionally anterior or posterior disc herniation can be seen on plane roentgenograms where a thin rim of calcification in the anterior or posterior longitudinal ligament demarcates the area of herniation. Spontaneous joint fusion occurs rarely.

Of the available diagnostic aids, the iodine contrast myelogram has proved the most useful. In our experience this study is safe, accurate and effective. Applicable to all patients, except the rare patient who is sensitive to iodine, it causes little morbidity other than occasional headache. Regardless of the usefulness of the myelogram, no patient should be subjected to surgery on the basis of myelographic findings only. Good plane roentgenograms of the lumbar spine must always be taken first. The practice of operating upon patients on the basis of myelographic findings alone is mentioned only to be condemned. The electromyogram, while occasionally helpful, has not in our experience proved nearly so reliable as the myelogram. Lumbar discography has not been used.

METHODS OF TREATMENT

One hundred ninety-five patients have been treated conservatively and 103 patients have been subjected to surgery. Patients examined in the early acute phase of their illness have been treated by adherence to the basic concept of rest. Confinement to bed at home or in the hospital, adhesive strapping to the lower back, and suitable medications for pain bring about relief of symptoms in three to ten days. Physical therapy including infra-red heat, massage and gentle stretching has been helpful. A firm bed and car seat are mandatory. The Goldthwait low back brace has proved to be a most effective aid in the conservative management of patients. This brace controls chronic symptoms with the exception of morning stiffness and it enables patients to engage in light physical exercise without fear of incapacitating attacks of pain. Obviously, the brace is no cure. However, some patients have worn their braces intermittently for as long as twenty years with satisfactory relief of symptoms. Ancillary therapeutic aids such as weight reduction and postural exercises have been used when indicated.

One hundred three patients were treated surgically. One patient was subjected to laminectomy only, and the remaining 102 were subjected to spinal fusion as well as laminectomy if exploration of the nerve root was indicated by preoperative signs and symptoms. Of these 103 patients, thirty-eight had disc herniations and nine had frank disc rupture with extrusion of nuclear material through a rent in the annulus fibrosis. The remainder had degeneration of the lumbar intervertebral joints of moderate to advanced degree with or without nerve root irritation due to foraminal compression.

FIG. 5. A lateral roentgenogram of a forty-eight year old woman taken ten years after fusion from the fourth lumbar vertebra to the sacrum. There is no evidence of significant degeneration above the fusion. The patient is asymptomatic.

The choice of patients for laminectomy and spinal fusion is important. Obviously, intractable sciatic pain from disc rupture requires surgical correction. However, when fusion is undertaken for relief of the symptoms of degeneration of lumbar joints the patient must understand that full cooperation is required during the convalescent period which can extend from six to twenty-four months. Unintelligent, willful, capricious, or psychoneurotic persons are not good risks for lumbar joint fusion. Patients with pending litigation, other than workmen covered by state compensation laws, are ill suited to spinal surgery for their subjective complaints are frequently far out of proportion to their objective findings.

Sixty-three lumbosacral fusions were performed. Twenty-nine patients were subjected to fusion from the fourth lumbar vertebra to the first sacral vertebra. (Fig. 5.) Six patients had fusions of the fourth lumbar joint only and two patients had fusions which extended from the third lumbar to the first sacral vertebra. The two remaining patients had segmental fusions of localized degenerative spaces leaving a normal joint between the fused portions of the lumbar spine.

The fusions have all been performed by a modified Hibbs technic. The paravertebral muscles have been meticulously reflected from the vertebral laminas by subperiosteal dissection. The ligamenta flava have been totally excised. The posterior zygoapophyseal joints have been eraded of cartilage and all synovial and capsular membranes excised. The defects caused are then filled by chips from the adjacent laminal arch. The laminas are then roughened, and the fusions have been completed by the addition of supplemental bone taken from an adjacent posterosuperior iliac spine through a separate incision. No support other than a firm adhesive strapping has been used in the immediate postoperative period. After sutures are removed on the tenth or twelfth postoperative day, the patients have been fitted with a back brace which is worn in bed until ambulation is started about the twenty-first day. Postoperatively, patients with fusions of one space have been kept in bed for a three-week period and thereafter have been gradually ambulated in a back brace. This is worn night and day for four months and during daytime hours until the fusion is solid by roentgenographic examination. Some patients with fusions of two or three spaces have been treated by a higher type of back brace. Very muscular male patients have been immobilized in a plaster cast for about eight weeks following surgery. Thereafter they have been fitted with a back brace. The integrity of all fusions has been tested by bending films taken nine months to two years after surgery.

In this small series there have been four failures of fusion, two at the lumbosacral level and two at the fourth lumbar level. Thirty-four patients have excellent results from their surgery in that they have obtained complete relief of preoperative complaints. Their physical examinations reveal no limitation of lumbar spine motion or other abnormal finding. Fifty-two patients have good results. They complain of occasional mild back or leg ache usually following physical exertion. Each of these patients has no less than 25 per cent loss of motion in the lumbar spine, especially extension and lateral flexion. Eight patients have fair results for although fusion was demonstrated on x-ray examination they complain of intermittent back and leg pain of varying

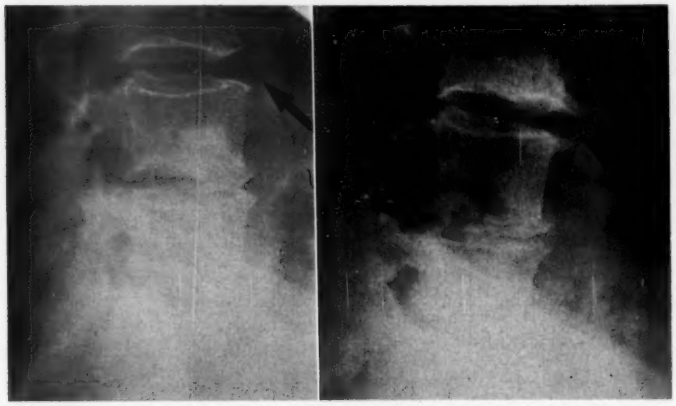


FIG. 6. Films taken over a five-year period to show the development of symptomatic degenerative disease in the disc space above the site of fusion in a fifty-eight year old woman in 1957. Surgery was performed in 1952.

degrees of severity. Eight patients have poor results. Four of these have compensation cases with pending claims and the other four failed to obtain surgical fusion.

COMPLICATIONS OF SURGERY

There have been no postoperative wound infections and no deaths. Acute parotitis developed in one patient due to a coagulase-positive Staphylococcus aureus which required surgical drainage. Bilateral phlebothrombosis developed in another patient which responded to therapy with anticoagulant drugs and supportive stockings. Symptomatic degenerative joint disease developed in seven patients above the site of fusion, six at the joint immediately adjacent and the other two joints above (Fig. 6.) Only one of these patients has been operated upon subsequently and laminectomy and fusion were performed for herniation of a third lumbar disc. He has a good postoperative result. The other six patients have been treated by conservative means, five with success and one without relief of pain.

Of special interest are sixteen patients in this study who were examined by me or my associates because previous surgery performed elsewhere had failed to relieve symptoms. Three patients had failure of fusion. In one patient the wrong intervertebral space was fused. The remaining twelve patients had been subjected to laminectomy with temporary or lasting relief of sciatic pain but with persistent low back complaints. (Fig. 7.) Careful study

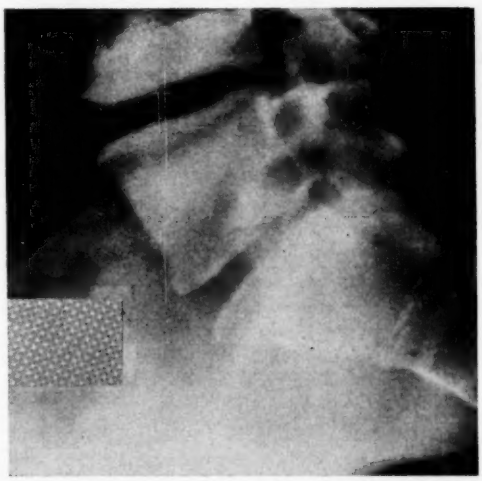


FIG. 7. Degeneration of the lumbosacral joint in a thirty-one year old man six years after he was subjected to laminectomy only. Back symptoms persist. The disc space below is transitional and does not move.

of their histories and roentgenograms reveals two facts. First, in the presence of roentgenographic evidence of degenerative lumbar joint disease laminectomy alone can relieve sciatic pain due to disc degeneration or rupture. Secondly, laminectomy alone will not relieve or alter symptoms in the lower back due to joint degeneration.

Although the patients subjected to surgery have been followed up for one to twenty years, no claim is made that their future shall be

free from back pain. However, bony fusion of one or more degenerated joints will relieve symptoms of the disease whether these are back or leg pain, or both. Surgical fusion moreover provides no assurance against further episodes of backache for many movable joints remain above the site of fusion. The over-all results from surgery in this relatively small group of patients have been most gratifying and as a group they lend further credence to the value of spinal fusion in the treatment of patients with degenerative lumbar joint disease.

SUMMARY AND CONCLUSIONS

Degenerative arthritis of the lower three lumbar intervertebral articulations is a chronic progressive disease which may span several decades of the patient's life. The lumbosacral joint, subjected to the greatest stress, is involved most frequently. Over two-thirds of the patients in this study have lumbosacral joint involvement only. While more common in the female, the sex differential is not significant. Symptoms may appear in the second decade of life. Symptoms develop in most patients prior to the age of forty. The onset of the disease can be acute or chronic. Acute attacks occur usually from simple body motions but are preceded by unrelated disease, chronic fatigue, emotional strain, infection, or trauma. Chronic symptoms are frequently underestimated by the physician, but these symptoms place real limitations upon the life of the patient. The collapse of the intervertebral disc space itself is slowly progressive over years not months, and there is no correlation between nerve root irritation and disc narrowing. The collapse of the disc causes derangement in the posterior zygoapophyseal articulations. In the early phase of the disease, acute attacks without disc herniation or rupture are self-limiting and the type of therapy makes little difference. Conservative treatment is effective in the control of both acute and chronic symptoms. Laminectomy alone, while able to relieve pain due to root irritation from disc herniation or rupture, in no sense affects the course of degenerating lumbar joints. Spinal fusion, however, with or without laminectomy can in the carefully selected patient relieve both the acute and chronic symptoms of this disease. The question of whether to fuse the involved portion of a patient's lumbar spine will be solved only through a better understanding of the pathologic anatomy of degenerative arthritis of lumbar intervertebral joints.

Low back pain with or without sciatic radiation is a common cause of human morbidity. So many people suffer from these symptoms that physicians of varying backgrounds are called upon to treat them. The internist, general surgeon, neurologic surgeon, industrial physician, and orthopedist must know something about the basic nature of degenerative changes which occur in lumbar intervertebral joints. In order to understand a disease, it is necessary first to record its natural history. This paper is presented as an effort toward that end.

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DISCUSSION

C. Hunter Sheldon (Pasadena, Calif.): There seems to be a great deal of controversy concerning the problem of low back and intervertebral disc syndrome from the orthopedic and neurosurgical point of view. Actually, there is no real controversy, because we are dealing with three different problems, the mechanics of the low back, sciatic pain, and the combination of both of these.

As for conservative and radical treatment, I think these are actually interchangeable, because if a person needs to be operated upon and is treated conservatively, that is radical treatment, and vice versa. I think any surgeon can remove a disc successfully, if he would reject those patients who should not be operated upon. This, I think, is the main problem, because if hemilaminectomy and removal of a disc are necessary, or if fusion is needed, and the proper procedure is selected, the results are going to be satisfactory. If the wrong

choice is made, you are fairly apt to have rather unsatisfactory postoperative results.

I often think that the decision as to what should be done depends upon the physician's experience.

I question the arthritis aspect of Dr. Wilson's report, because I certainly think no inflammatory process is involved. The changes which occur around the joints are probably not arthritic but the result of altered mechanics. The disc space, of course, is largely fluid. If the disc were dehydrated you would have little material left. And because it is largely water it must function on a hydraulic principle. So that when you bend the spine forward, backward, or sideways, the pressure at each point along the surfaces is equal.

It seems to me that during one's lifetime some type of situation other than arthritis must occur to account for the occurrence of these lesions; I think that the changes are probably chemical. Maybe they have to do with some endocrine or dietary process. Certain things I believe are fairly well established, one of which is that if the lesions were purely mechanical from lifting or twisting, these would occur primarily in the teen-agers who lift anything, or attempt to lift anything, that is loose. Yet they seldom occur at that age as compared with the thirties, forties and fifties.

Some years ago, when at Bethesda, Dr. Barr of Boston and I attempted to produce rupture of discs by mechanical pressure. We had fresh spines and put cups of various types around the vertebrae above and below and subjected them to tremendous degrees of pressure. We never were able to rupture a disc. We could crush the vertebra above and below, that is, the body, but never rupture the disc. In fact, we had to drill holes of fairly good size into the discs to get anything to come out.

It has been shown that there are certain chemicals within the disc; hyluronic acid is one of them. Its role is uncertain, but in people with degenerative discs this chemical is very much reduced in amount.

There is an interesting condition called lathyrism which occurs in animals fed sweet peas, in which a marked degeneration of the discs and changes in the cartilaginous structure occur. It would seem to me that probably the combination of chemical changes, possibly dietary changes, and later the superimposing of trauma on the discs may account for the ruptures.

This disc, if observed in a cross-section cut, has a soft center called the nucleus pulposus, which is the remnant of the notochord structure. Around it are concentric rings or laminated layers which become tougher and tougher as one approaches the periphery. I have envisioned this process as one in which, due to some force, a few of the innermost laminated layers are ruptured, and the central gelatinous nucleus extrudes itself through the torn edges of the laminated layers. As a result acute pain or strain develops, and this results in unlocalized pain which is more or less in the low back without any sciatic radiation.

Three or four or possibly a hundred of these may rupture, followed by an acute recession of symptoms as the edema and reaction disappear. Each time something is lifted, this ameboid projection of nucleus pulposus extends out through a few more laminated layers, producing at each episode lumbago, low back pain, or sacroiliac symptoms. It is not until you have a fairly extensive migration of this material that you begin to encroach upon the structures near the periphery and at this point begin to irritate the nerve root. The pain then radiates into the buttocks and maybe the upper thigh.

As this occurs, the disc dehydrates, loses its water content, and now functions on the mechanical principle. When one bends forward the pressure is far greater anteriorly than posteriorly. The disc migration tends to progress posteriorly, and nature, as it often does elsewhere in the body, reacts because of excessive pressures, and then hypertrophic changes occur which always are predominately anterior as compared with posterior.

When this ruptures, finally, to the intraspinal space, there is a cessation of back pain and a marked exaggeration of sciatic pain.

During the intermediate stages, as I previously mentioned, both low back and sciatic pain are present.

As to what ought to be done once it is partially or completely extruded, depends upon one's point of view. From our standpoint the most important diagnostic features are the positive neurologic findings. These may be weakness of the big toe, absent Achilles reflex, or some sensory changes. The importance of these is not to signify that the nerve is being irritated, because the sciatic pain will demonstrate to the patient as well as to the physician that this is occurring, but if the pain suddenly subsides in a patient who has marked sciatic pain with some neurologic findings, frequently they are considered improving. However, if at the same time the neurologic signs increase as the pain decreases, the patients are subjectively better, but potentially from the standpoint of damage to the cauda equina they have progressed considerably to a much more precarious situation. This alone may be enough justification for operation.

If one is going to adequately arrive at the proper solution, neurologic examination, electromyography, and myelograms should all be performed, to reduce the possibility of error. The myelogram will also exclude the presence of a tumor of the cauda equina at a higher level, which may be present in 1 to 3 per cent of persons.

JOHN C. WILSON, JR. (closing): I think the

author of any essay on backache is likely to find himself making a series of positive statements, and, as you know, there is harm in being always positive, for you may not be always right.

However, the conclusions which I have presented to you have been formulated after careful study. The diagnostic criteria and roentgenologic interpretations are founded upon a firm belief that degenerative disease in lumbar vertebral joints, and especially in the lumbosacral joint, is the major cause of chronic recurrence of severe low back pain.

Today, in industrial medicine, there is no problem which is handled less well than the patient with the injured lower back, with or without sciatica. Low back pain, with or without sciatic radiation, is, as you know, a common cause of human morbidity. So many people suffer from these symptoms that physicians of varying backgrounds must of necessity treat them. The internist, general surgeon, neurologic surgeon, industrial physician, as well as the orthopedist, must know something of the basic nature of the changes which occur in lumbar joints.

However, I think in order to understand this disease it is necessary first to record its natural history, and the present study has been an effort toward that end.

Tumors of the Deep Lobe of the Parotid Gland

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THE vast majority of tumors of the parotid occupy that portion of the gland which lies superficial to the facial nerve and its branches. The surgical management of these tumors has received wide attention and a fairly standard approach with exposure and protection of the nerve and removal of the entire superficial portion of the gland with the tumor has been accepted. Tumors lying deep to the plane of the facial nerve present a more difficult problem in both diagnosis and therapy. This report details our experience with benign tumors of the deep lobe of the parotid gland.*

A total of eight patients with benign parotid tumors lying deep to the plane of the facial nerve have been encountered on the surgical services of the Veterans Administration Hospital, Portland, Oregon, and in private practice between the years 1950 to 1959. These tumors have been representative of the varying anatomical locations, clinical presentations, and surgical problems which may be encountered in benign tumors of the deep lobe of the parotid. Malignant tumors involving the deep lobe have not been included in this study since their management has not generally required the maneuvers to preserve the facial nerve that we wish to emphasize.

Anatomy. The greatest mass of the parotid (superficial lobe) lies molded over the masseter muscle and the angle of the mandible, extending posteriorly to the mastoid process and the anterior border of the sternocleidomastoid muscle. It overlies the facial nerve which penetrates the parotid at its posterior border and divides within the gland. Beneath the facial nerve and its branches, an additional thin layer of parotid tissue may extend forward a short distance over the masseter muscle in a relatively superficial position. The bulk of the deep portion of the parotid, however, curves around the posterior border of the ramus of the mandible filling the gap between the mandible and the mastoid process. (Fig. 1.) The deepest aspect of the parotid, the so-called retromandibular process, occupies the lateral pharyngeal (pterygomaxillary) space. This space is bounded medially by the pharyngeal wall. The anterolateral boundary is formed by the internal pterygoid muscle and the ramus of the mandible. Posteriorly the space is separated from the internal carotid artery and the internal jugular vein by the styloid process and the muscles originating from it. The lateral pharyngeal space extends superiorly to the tympanic plate at the base of the skull, while inferiorly the broad stylomandibular ligament and fascia separates it from the posterior extremity of the submaxillary salivary gland.

The deep lobe of the parotid is traversed by the posterior facial vein and its main branches all of which lie deep to the facial nerve. The external carotid artery also ascends through this portion of the gland and divides into its two terminal branches. The amount of parotid tissue which lies deep to the facial nerve varies widely. It may consist in merely a small button of tissue beneath the main trunk of the nerve with a very thinned out retromandibular process, or it may be almost equal in size to the superficial portion of the gland.

* Although a number of excellent studies [6,10] have established the fact that the parotid is a distinctly unilobar gland, we have continued to employ the terms "superficial lobe" and "deep lobe" in designating portions of the gland which lie on either side of the plane of the facial nerve. While these terms may be anatomically incorrect, it is believed that for clinical purposes they are useful and are understood by all surgeons. Furthermore, the term "lobectomy" or "superficial lobectomy" when applied to an operation for parotid tumor implies a far more specific procedure than does the expression "wide removal" or "subtotal parotidectomy."

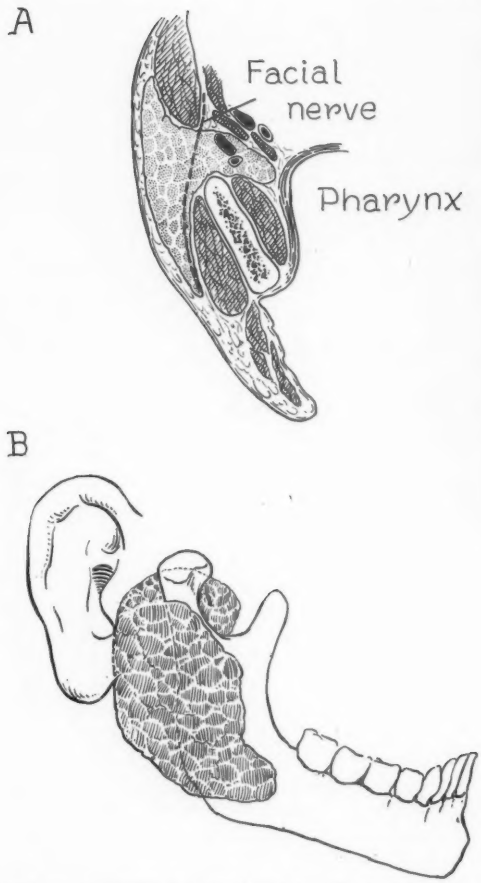

FIG. 1. Anatomy of the deep lobe of the parotid. A small thinned out portion of the deep lobe may extend forward over the masseter muscle beneath the facial nerve. The remainder fills the gap between the mastoid process and the mandible and extends medially into the lateral pharyngeal space.

Symptomatology and Diagnosis. Parotid tumors lying beneath the facial nerve may present clinically in one of three different ways depending on the site of the tumor in the deep portion of the gland: (1) the tumor may bulge outward in the upper neck and over the angle of the mandible; (2) the tumor may present itself only in the pharynx; and (3) the tumor may be visible and palpable both in the pharyngeal wall and externally in the neck.

Those neoplasms which present externally cannot be distinguished clinically from the common benign tumors of the superficial lobe of the gland. (Fig. 2.) There is generally a long history of a painless lump beneath the lobe of the ear which has slowly increased in size. Although the facial nerve may become markedly stretched and distorted in its course over the external surface of the tumor no facial weakness has been observed. Despite the fact that tumors in this location are covered by the superficial portion of the parotid they may seem to lie just beneath the skin on palpation and may be freely movable. In only one of our patients in whom the tumor presented externally only, did the tumor seem fixed to deeper structures on examination.

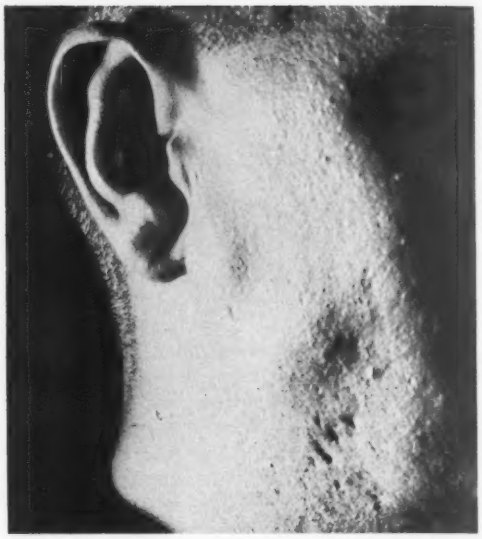

FIG. 2. Case 1. Deep lobe tumor of the parotid gland. Inspection and palpation suggested that this parotid mass lay just beneath the skin. At operation the tumor was confined to the deep lobe beneath branches of the facial nerve.

The retromanidbular tumors which bulge into the pharynx are the most interesting of the deep lobe parotid neoplasms. They are encountered as firm, rounded masses in the lateral pharyngeal wall covered by intact mucosa. They usually present superior to the tonsil and extend outward and upward into the soft palate. In one patient the tumor presented mainly in the lateral wall of the nasopharynx causing only slight distortion of the soft palate which could easily be missed on intraoral examination.

The usual complaint of a patient with a retromandibular parotid tumor is of a sense of fullness and discomfort in the throat. There may be slight dysphagia and change in the voice. In the one patient whose tumor presented in the nasopharynx the most distressing symptom was unilateral deafness. It is likely that tumors in this site exist for long periods of time without causing symptoms. When our patients finally became aware of the tumor and sought medical aid the tumors were already quite bulky, one even bulging into the pharynx as far as the midline. Despite the size these tumors have reached there has been no ulceration of the overlying mucosa.

The diagnosis of retromandibular parotid tumor may be quite obscure to those not familar with this form of presentation of parotid neoplasms, particularly when no external component is palpable in the neck. In one of our patients the pharyngeal mass was thought to represent a peritonsillar abscess and was incised twice in a fruitless search for pus. In another patient, even after a biopsy through the pharynx revealed benign mixed tumor, the mass was thought to represent a tumor of ectopic salivary tissue rather than one arising in the parotid gland. On rare occasions various neurogenic tumors may have a similar clinical presentation. In our experience, however, a firm tumor mass in the upper lateral pharyngeal wall covered by intact mucosa must be considered a tumor of the deep lobe of the parotid until proved otherwise. Bimanual palpation through the pharyngeal wall and upper neck under local or general anesthesia has been an aid to diagnosis. (Fig. 3.) By this maneuver the continuity of the tumor with the more superficial portions of the parotid gland may be established. We have not obtained sialograms on any of the patients with retromandibular parotid tumors. It seems likely that the bulk of the tumor would so compress the ducts in the deep lobe of the gland that no filling could be obtained.

Diagnosis is less difficult when the tumor presents both externally and in the pharyngeal wall. The continuity of the two masses is easily recognized on bimanual palpation. In one patient the external component of the tumor appeared beneath the body of the mandible, as close to the submaxillary salivary gland as to the superficial portion of the parotid, making its parotid origin less obvious. More typically the external mass lies beneath the lobe of the ear, clearly within the confines of the parotid gland.

Treatment. The management of tumors of the superficial lobe of the parotid has been described in detail by numerous authors. Most surgeons interested in parotid neoplasms have accepted the principle of preliminary exposure of the facial nerve followed by wide removal of the tumor with a surrounding margin of normal parotid tissue. This procedure usually includes excision of the entire superficial lobe of the parotid [*4,11*]. Some authors have gone a step further and advocated total parotidectomy for all benign mixed tumors [*3,9*]. A catastrophe awaits the surgeon who mistakenly attempts to enucleate a deep lobe tumor bulging outward in the neck without first identifying the facial nerve. These tumors so distort the nerve and the superficial portion of the gland that one or more divisions of the nerve are very likely to be severed before the surgeon is aware of the unusual situation.

The surgeon experienced in parotid procedures who first identifies the facial nerve will recognize the deep lobe localization of the tumor as the operation proceeds. The standard dissection is continued, removing the superficial lobe of the gland and exposing all branches of the nerve which are seen to be stretched out over the tumor. No attempt should be made to remove the superficial and deep portions of the gland in continuity.

When removal of the superficial lobe is complete, careful evaluation of the relation of the facial nerve and its branches to the underlying tumor will help determine the next step. Superficially located tumors of the deep lobe tend to lie anteriorly over the ramus of the mandible and the masseter and may be delivered outward between branches of the nerve. (Fig. 4.) At times, however, the main trunk of the nerve and all its branches must be elevated and retracted superiorly allowing the tumor to be pulled downward for removal. When the method of removal was been decided upon, the facial nerve and its branches are carefully dissected away from the underlying tumor. To avoid incision of the tumor, dissection must generally be carried immediately beneath each branch of the nerve, gently elevating it with fine nerve hooks. When the nerve has been entirely disengaged, the branches are separated or the entire nerve

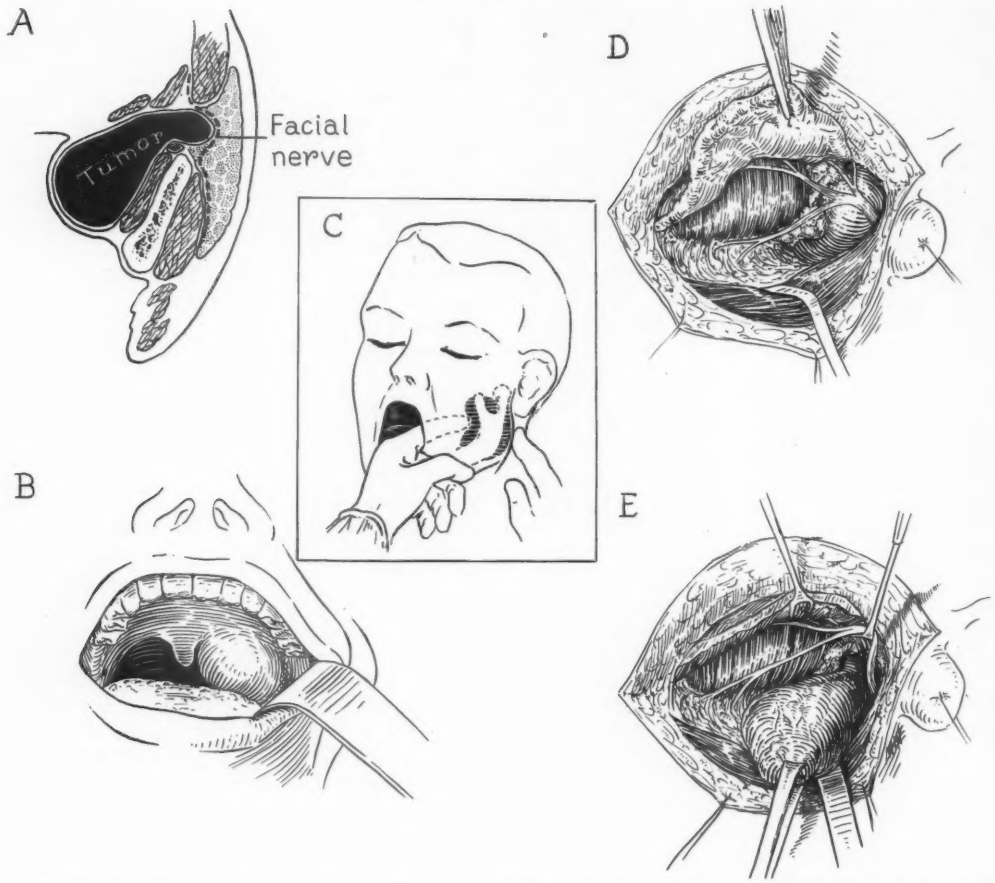

FIG. 3. Deep lobe tumor of the parotid gland. A, a tumor of the retromandibular portion of the deep lobe bulges into the lateral pharyngeal wall and may or may not present externally in the neck. B, usual appearance of the pharyngeal mass. C, bimanual palpation is an aid in demonstrating the parotid origin of the pharyngeal mass. D, superficial lobectomy is being performed with exposure of the outer aspect of the deep lobe tumor. The facial nerve may be markedly displaced and stretched by the mass. E, the facial nerve is retracted superiorly allowing the tumor to be delivered downward into the neck.

retracted upward to allow removal of the tumor.

The management of retromandibular parotid tumors may be more difficult, and their removal has taxed the ingenuity of surgeons. Erlich [2] has described a transoral approach with incision of the overlying mucosa and dissection of the lateral pharyngeal space. Morfit [7], in reporting on twelve patients with benign and malignant retromandibular parotid tumors, advocated a submandibular approach with removal of the submaxillary salivary gland. The tumor is then freed by blunt dissection and delivered downward beneath the body of the mandible. Ariel, Jerome and Pack [1] have recommended a more direct approach, performing a standard parotid lobectomy and then transecting the ramus of the mandible or removing a block of bone from its posterior portion for exposure of the deep lobe tumor. Ward and Hendrick [8] and Martin [5] also utilize the standard parotid lobectomy and describe delivery and removal of the tumor without transection or partial removal of the

mandible. We have found that retromandibular tumors may be removed with little difficulty using the technic described. (Fig. 3.)

A standard incision for surgery of the parotid is made, the facial nerve identified and the superficial portion of the gland is removed. The main trunk of the nerve and its divisions are gently dissected away from the underlying tumor. The nerve and its main branches must be elevated superiorly, since the bulky retromandibular tumor must be delivered downward into the neck. At times, knoblike projections of the tumor will hinder attempts to retract the nerve superiorly. The tumor mass must then be pushed inward toward the pharynx to allow complete upward mobilization of the nerve. The external carotid artery is divided and ligated just below its entrance into the deep portion of the parotid.

The working space between the ramus of the mandible and the mastoid process may be widened by sliding the mandible forward into a prognathic position. While this maneuver helps in mobilization of the tumor, this space has not been adequate for delivery of the bulky retromandibular masses we have encountered. For removal, the tumor must be dislocated downward into the neck.

Blunt finger dissection is generally effective in separating the retromandibular tumor from the pterygoid muscles and from the pharyngeal wall. The tumor is then pulled downward into the upper neck while the facial nerve and its branches are gently retracted upward. An assistant's finger placed in the patient's pharynx making downward pressure on the tumor is of aid in delivery of the mass. After delivery of the tumor, bleeding from the lateral pharyngeal space is usually minor and is not difficult to control. A drain is placed deep to the ramus of the mandible and the wound closed in the usual manner. We performed a prophylactic tracheostomy in our first patient with a retromandibular parotid tumor but encountered no postoperative impairment of the airway and have not used a tracheostomy since.

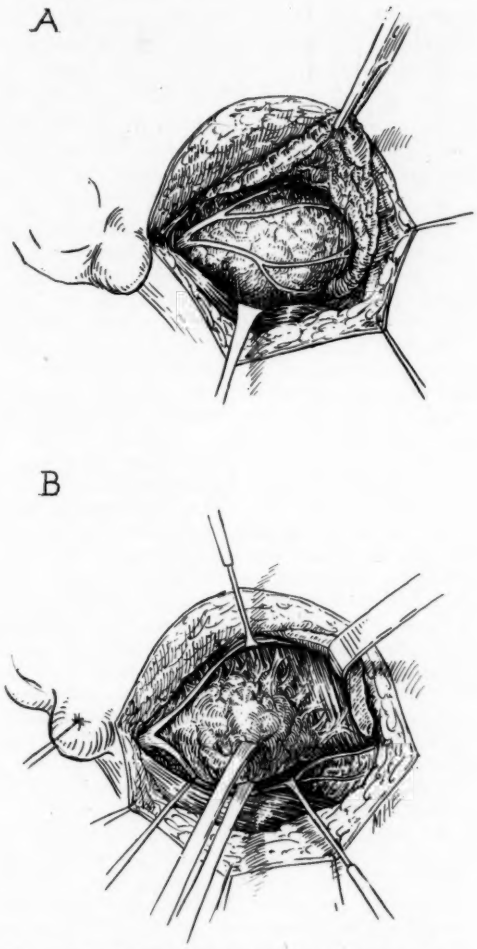

FIG. 4. Deep lobe tumor of the parotid gland. A, superficial lobectomy is being completed with exposure of a tumor of the superficial portion of the deep lobe. B, branches of the facial nerve are retracted apart allowing the deep lobe tumor to be delivered between them.

CASE REPORTS

A total of eight benign tumors involving the deep lobe have been encountered between 1950 and 1959 in the surgical treatment of over 100 benign parotid lesions. Pathologically, four of the neoplasms were primary benign mixed tumors, two were Warthin's tumors, and two were recurrent mixed tumors appearing after simple enucleation of tumors of the superficial portion of the gland performed elsewhere.

All of the deep lobe tumors were removed in the manner described with preservation of the facial nerve and its branches. Because of the extra manipulation of the nerve required for removal of the deep lobe of the parotid, transient facial weakness following surgery is much more common than that seen after routine superficial parotid lobectomy. Six

patients had paralysis of facial muscles following operation; the facial palsy was complete in two, in three it involved only the lower lip, and in one the upper part of the face. Complete recovery of function occurred in all patients. All eight patients have been followed up since operation without evidence of recurrent tumor.

CASE I. A. S., a thirty-two year old man, was admitted to the Portland Veterans Administration Hospital in September 1952 complaining of a tumor anterior to his right ear. The mass had been present for many years but had recently become enlarged. Examination disclosed a very firm, well circumscribed, 3 by 2 cm. non-tender mass in the right parotid region which seemed to lie just beneath the skin. The mass was only slightly mobile; the facial nerve was intact.

Operation was performed on September 9, 1952. As the facial nerve was exposed and mobilization of the superficial lobe commenced, it was discovered that the tumor originated in the deep portion of the gland, bulging outward and separating the zygomatic and buccal branches of the nerve. The superficial lobe of the parotid was removed except for a small peripheral rim of tissue. The branches of the facial nerve were then dissected away from the tumor and the deep lobe of the gland containing the tumor was delivered between the buccal and zygomatic branches of the nerve. The pathologic report was benign mixed tumor of the deep portion of the parotid gland.

Following surgery the patient had weakness of muscles innervated by the upper divisions of the facial nerve. There was full recovery by March 1953. Gustatory sweating (Frey syndrome) in the operative site has persisted. The patient was last examined in December 1959 and no evidence of recurrent tumor found.

CASE II. P. Q., a twenty-six year old man, had a small mixed tumor of the left parotid gland enucleated in 1943. A recurrent nodule was locally excised in 1946 in the Navy. Another nodule appeared in 1949 and the patient was admitted to the Portland Veterans Administration Hospital in November 1950. Examination revealed an irregular, firm, 4 cm. mass in the left parotid region fixed to the underlying tissues. There were two healed incisions in the skin each less than 3 cm. in length. The facial nerve was intact.

At operation on November 22, 1950 the superficial portion of the parotid containing several small tumor nodules was removed with isolation and protection of the facial nerve. The bulk of the tumor occupied the deep lobe which was removed after the main trunk of the nerve and its branches had been mobilized and retracted superiorly. Examination of the specimen revealed multiple nodules of benign mixed tumor in both the superficial and deep portions of the gland. There was weakness of all muscles innervated by the facial nerve following surgery. Complete recovery was apparent by February 1951. The patient was last seen in March 1959 without evidence of recurrence.

CASE III. C. C. noted a small mass overlying the left angle of the mandible in 1945. A biopsy specimen was obtained at that time in a Navy hospital. Pathologic examination revealed a 7 mm. fragment of normal parotid tissue. The patient stated the mass persisted and gradually increased in size until January 1952 when he was admitted to the Portland Veterans Administration Hospital at twenty-five years of age. Examination revealed a firm, movable, 3 by 4 cm. mass beneath the lobe of the left ear and overlying the angle of the mandible. There was a small healed scar in the skin over the mass.

Following exposure of the facial nerve on January 9, 1952, the tumor was discovered to occupy the deep lobe of the parotid. The inferior three-fourths of the superficial lobe was resected, the facial nerve retracted upward, and the entire deep lobe of the gland containing the tumor was removed. Microscopic examination revealed a Warthin's tumor. There was weakness of the lower lip following surgery which disappeared within three months. There was no sign of recurrence on re-examination in March 1959.

CASE IV. J. W., a forty-five year old man, was admitted to the hospital in April 1956 with the chief complaint of recurrent parotid tumor. A benign mixed tumor of the right parotid was enucleated in an Army hospital in 1944. A recurrent nodule appeared in 1953 and slowly increased in size. On examination a 3 by 4 cm. irregular mass was palpable immediately beneath a small vertical scar underlying the lobe of the right ear. There was no cervical adenopathy and no facial weakness.

At operation on April 11, 1956, a tumor involving the remnants of the superficial lobe of the parotid was found to extend directly into the deep portion of the gland. A superficial lobectomy was performed transecting a portion of the tumor. The facial nerve was freed from the underlying tumor and the greater portion of the deep lobe of the gland was delivered between the zygomatic and buccal branches of the nerve. The pathologic report was benign mixed tumor. There was weakness of muscles innervated by the buccal and mandibular branches of the facial nerve following surgery with complete recovery by July 1956. The patient was re-examined in March 1959 and no evidence of recurrence discovered.

CASE V. L. H., a fifty-nine year old man, was admitted to the hospital in December 1957 complaining of bilateral painless swelling of the parotid gland. A mass had appeared in the left parotid region five years prior to admission, and in the right parotid region two years prior to admission; each had slowly increased in size. Examination disclosed a firm, well circumscribed 4 by 6 cm. mass overlying the angle of the mandible on the left; a 3 cm. mass was present on the right just anterior to the mastoid process. The preoperative diagnosis was bilateral parotid neoplasm, probably Warthin's tumors.

The larger left parotid mass was operated upon on January 14, 1957. The superficial lobe of the parotid was removed exposing a tumor of the deep lobe lying beneath all branches of the facial nerve. The buccal branch of the nerve was elevated and the cervical and mandibular branches retracted downward allowing delivery and excision of the tumor. The pathologic report was Warthin's tumor. There was no facial weakness following surgery. One month later a parotidectomy was performed on the right side for a Warthin's tumor of the superficial lobe. The patient was examined in April 1959 and found to be without evidence of recurrence.

CASE VI. V. V. F., a fifty-seven year old man, first became aware of discomfort in his throat in 1947. He was found to have a mass bulging into the left side of the pharynx in February 1949 at which time an excision through the mouth was attempted but complete removal was impossible. Intensive external radiation through anterior, lateral, and posterior oblique fields was therefore given. The tumor slowly increased in size and the patient was admitted to the Portland Veterans Administration Hospital in September 1954.

Examination disclosed a 7 by 5 cm. mass filling the left side of the pharynx and extending almost to the midline. The overlying mucosa was intact. There were extensive radiation changes in the skin over the parotid but no external mass was palpable. There was no cervical adenopathy. Bimanual palpation suggested continuity of the pharyngeal mass with the parotid gland.

At operation on October 4, 1954, there was marked scarring of the superficial lobe of the parotid gland which was separated from the facial nerve with difficulty. The nerve was then mobilized and elevated and the external carotid artery divided between ligatures. With external traction and blunt dissection as well as pressure from within the mouth, the large tumor mass was delivered downward into the neck. A temporary tracheostomy was performed at the conclusion of the operation. Examination of the specimen revealed a benign mixed tumor of the deep lobe of the parotid 6 cm. in greatest diameter. Wound healing was delayed due to radiation damage of the skin. There was complete postoperative facial palsy which greatly improved by December 1954 and disappeared by June 1955. There was no evidence of recurrence on recheck examination in September 1959.

CASE VII. N. C., a forty-three year old married woman, noted a painful mass in her throat following an upper respiratory infection in February 1958. There was no improvement after two weeks of antibiotic therapy so she was admitted to the hospital with a diagnosis of peritonsillar abscess. Incision and drainage of a mass in the pharynx was attempted under local anesthesia but no pus was obtained. After several days of intensive antibiotic therapy, incision and drainage was again attempted without success. A mass palpable beneath the body of the mandible was then biopsied with a pathology report of possible low grade fibrosarcoma.

Examination on March 29, 1958, revealed a firm, tender, 4 by 5 cm. submucosal mass bulging outward from the lateral pharyngeal wall and displacing the right side of the soft palate. The overlying mucosa was reddened and there were two recent incisions which were healing. There was induration and a poorly defined mass palpable beneath the posterior third of the body of the mandible. Bimanual palpation demonstrated continuity of the pharyngeal and submandibular masses and a diagnosis of tumor of the deep lobe of the parotid was made.

Operation was carried out on March 31, 1958, by Dr. Edward E. Wayson, assisted by one of us. The main trunk of the facial nerve was identified and the superficial lobe of the parotid removed. The nerve and its branches were then elevated and a bulky retromandibular mass mobilized by blunt dissection. With external traction and digital pressure within the pharynx, the tumor was delivered downward into the neck. On histologic study of the entire specimen the diagnosis was changed to benign mixed tumor. The patient had weakness over the mandibular distribution of the facial nerve at the time of discharge from the hospital on April 8, 1958. There was partial recovery by June 1958. She was last examined in April 1959 and no evidence of recurrent tumor noted.

CASE VIII. M. S., a fifty-eight year old married woman, first noted a nodule in the right parotid region in 1956. She consulted an otolaryngologist in January 1959 because of deafness in the right ear. He discovered and biopsied a nasopharyngeal mass with a pathologic report of benign mixed tumor. Examination of the neck disclosed a firm, fixed mass filling the space between the mastoid

process and the angle of the mandible and elevating the lobe of the ear. Facial nerve function was normal. Intraoral examination revealed only minimal depression of the right side of the soft palate. Inspection of the nasopharynx revealed a bulky submucosal mass filling the entire right side of the nasopharynx and apparently arising from the right nasopharyngeal wall. On bimanual palpation the nasopharyngeal mass could be moved in continuity with the mass in the neck.

At operation on April 20, 1959, the superficial lobe of the parotid was normal and was entirely removed. The facial nerve was markedly stretched downward and outward by the external bulge of the tumor mass; the main trunk of the nerve measured 4 cm. in its course from the stylomastoid foramen to its point of division. It was necessary to force the tumor inward in order to loop the nerve superiorly over the projecting knob of tumor. The external carotid artery was then divided. The inner aspect of the tumor was mobilized by blunt dissection and the entire mass delivered downward into the neck. The specimen was a dumbbell-shaped mass 3 by 7 cm. in greatest dimensions which on histologic examination proved to be a benign mixed tumor. The patient was discharged from the hospital on April 19, 1959, with normal hearing and no facial weakness. She was re-examined in December 1959 with no evidence of recurrence.

SUMMARY

1. The anatomy of the deep portion of the parotid and "deep lobe tumors" is reviewed.

2. The symptomatology and diagnosis of benign tumors of the deep lobe of the parotid is described.

3. Surgical treatment consists in removal of the superficial lobe of the parotid followed by retraction and preservation of the facial nerve and removal of the deep lobe containing the tumor.

4. Eight patients with benign tumors of the deep lobe of the parotid are reported on. All tumors were successfully removed without permanent facial nerve damage and without recurrence to date.

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DISCUSSION

William B. Hutchinson (Seattle, Wash.): The work that Drs. Wise and Baker have presented is evidence of extensive experience with parotid tumors. The so-called superficial lobe tumors in our series have occurred seven or eight times more often than the deep lobe tumors. The segregation of the two lobes of the parotid on a clinical basis carried out by the authors at operation seems justifiable to me, in spite of certain criticisms by the anatomical purists.

I should like to discuss the case of an eighty-one year old woman that illustrates some of the points mentioned by Dr. Baker. She was rejected for surgery by me about eleven years ago because of a very marked hypertension. She has survived to see her tumor reach tremendous proportions, but the tumor actually is asymptomatic. This would have been a difficult tumor to remove at any stage of growth after her first visit. In removing a deep lobe tumor it has always seemed to me that when the dissection has begun at the periphery and moved inward towards the main trunk of the facial nerve, that the situation as regards the site of origin of the tumor and the safety of the main trunk and branches of the facial nerve is much more certain. Authors have described in detail a satisfactory method of removing deep tumors of the parotid but a particularly large tumor will be a formidable task if one preserves the nerve and does not enter the tumor.

In any mixed tumor or moderate sized carcinoma, removal of the entire parotid gland with preservation of the facial nerve is the preferred method of treatment. Enucleation, if practiced routinely, will lead to a very high percentage of recurrences. We know from treating a group of forty patients, all followed up over ten years and in some instances for as long as seventeen years, that this problem of recurrence is non-existent when the patient is treated by radical parotidectomy with preservation of the facial nerve. This includes the tumors of the deep lobe as well.

HARVEY W. BAKER (closing): I would like to thank Dr. Hutchinson for his kind comments. We have been impressed with and have admired his work in the treatment of parotid tumors.

He is certainly in good company when he advocates a total parotidectomy for even benign tumors of the superficial lobe. The late Dr. Grant Ward and his group at Johns Hopkins made similar recommendations.

We have still not been impelled to remove the deep lobe of the parotid for tumors involving the superficial lobe when we have been able to perform a superficial lobectomy without getting into the tumor or without getting too close to it; so far there have been no recurrences in such patients. We have been impressed by Dr. Hutchinson's series of patients whom he carefully followed up for over ten years; we are going to be interested in follow-up studies in our own patients as time goes by.

Deep Hypothermia for Intracardiac Surgery

EXPERIMENTAL AND CLINICAL USE WITHOUT AN OXYGENATOR

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THE ultimate goal for intracardiac surgery must be the simplest, safest and most efficient technic for operating within the chambers of the heart without jeopardizing the welfare of the patient or compromising the ability of the surgeon to accomplish the best possible repair. Extracorporeal equipment should be compact and uncomplicated and require the least possible amount of blood for priming. The operative field should be quiet and bloodless without the use of drugs or anoxia, and perfusion should be accomplished without significant hemolysis, acidosis or imbalance of blood volume.

It appears to us that deep hypothermia without an oxygenator best satisfies these requirements at the present time. We have used a system similar to that of Drew and associates in England [1,2] and Shields and Lewis in this country [3] to study this method in the laboratory. The success of this method has prompted us to adopt it in treating a series of patients with congenital and acquired cardiac lesions requiring open heart surgery.

This report details our animal studies and the results of the initial series of patients operated on under deep hypothermia without an oxygenator.

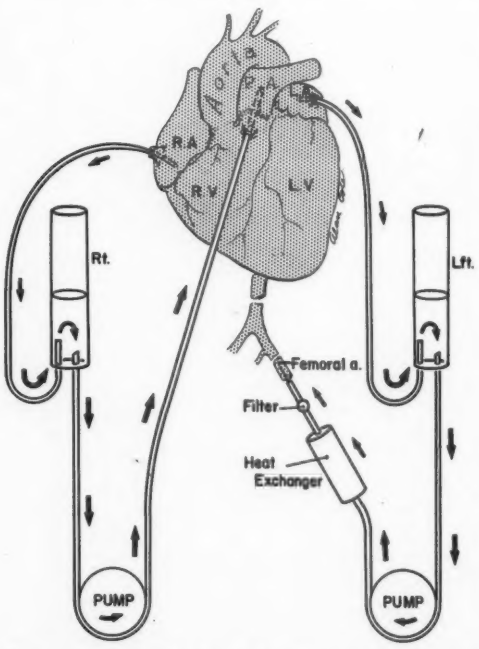

FIG. 1. Diagram showing basic set-up and equipment for deep hypothermia.

BASIC TECHNIC FOR DEEP HYPOTHERMIA

The basic set-up and equipment have been the same for the animal studies and the clinical cases. As diagrammed in Figure 1, the extracorporeal circuit involves gravity drainage of the right atrium into a reservoir from which the venous blood is pumped to the pulmonary artery. Blood is also drained by gravity from the left atrium into a reservoir from which it is pumped through a heat exchanger and filter to the femoral artery. Occlusive roller-type pumps have been used and the heat exchanger is the Brown-Harrison model developed at Duke University [4]. For cooling, the heat exchanger has been used in conjunction with a recirculation bucket containing crushed ice and water which maintains a temperature of between 0° and 2°C. For rewarming, hot and cold tap water have been used and maintained

at a temperature of 40°C. by the heat exchanger regulator.

ANIMAL STUDIES

Procedure. Thirty mongrel dogs ranging in weight from 14 to 46 kg. have been used for the animal studies. No preanesthetic medication was given and anesthesia was induced and maintained with Fluothane.® Pressure in the femoral artery was measured continuously by means of a strain gauge. Lead II electrocardiograms were recorded from subcutaneous needle electrodes and the esophageal temperature was monitored by means of a Tele-Thermometer.®

The chest was entered through a sternal splitting incision and catheters were placed in the atria, pulmonary artery and femoral artery after heparin was administered in doses of 3 mg. per kg. of body weight. The animals were then cooled and subjected to varying periods of circulatory arrest. Right ventriculotomy was performed on all except the first five animals. After rewarming to near normal body temperatures, the catheters were removed and Polybrene® administered in one and a half times the amount of heparin. The chest was closed with intercostal drainage tubes in both sides. Postoperative treatment routinely included the administration of intramuscular penicillin, demerol for obvious pain and intermittent positive pressure breathing when indicated. Chest tubes were removed after twelve to forty-eight hours when there was no further evidence of significant drainage.

Results. The over-all results are summarized in Table 1. All except three of the animals survived the procedure. The deaths occurred among the first twelve dogs operated upon. The first animal in which the procedure was performed died from air emboli that resulted from faulty equipment. Another dog died due to fulminating pulmonary edema from overtransfusion. The third who died was an old police dog weighing 100 pounds who was used to test the effectiveness of cooling a large animal. At surgery he was found to have an occlusion of one of the major coronary arteries. He tolerated the surgical procedure but it was impossible to maintain an effective cardiac output after rewarming. All other animals survived the procedure without evidence of neurological or other sequelae. Many of the animals showed weakness of the hind legs of about one day's duration. The cause of this was not determined, but controlled tests on other dogs indicated that it was not due to ligation of both femoral arteries after their use during surgery or to the effects of the anesthesia. All animals so afflicted regained use of their hind legs within a day or two.

TABLE 1
MEAN VALUES FOR THIRTY DOGS UNDER DEEP HYPOTHERMIA

Data	Group A (22 dogs cooled to 13° to 18°C.)	Group B (8 dogs cooled to 8° to 12°C.)
Cooling		
From	36°C.	35°C.
To	15°C.	10°C.
Temperature drop	21°C.	25°C.
Time for drop	21 min.	30 min.
Rate of drop	1°C. per min.	1°C. per 1.2 min.
Circulatory arrest		
Time	25 to 35 min.	55 to 60 min.
Temperature drift up	2° to 3°C.	5° to 7°C.
Rewarming		
From	18°C.	15°C.
To	35°C.	34°C.
Temperature rise	17°C.	19°C.
Time for rise	24 min.	28 min.
Rate of rise	1°C. per 1.4 min.	1°C. per 1.5 min.
Survival	20	7
Incidence of ventricular fibrillation	19	8

All except three of the dogs went into ventricular fibrillation at some time during the procedure. Most of them fibrillated between 14° and 22°C. Those that did not fibrillate by the time the temperature reached 14°C. went into cardiac arrest but fibrillated when ventriculotomy was performed or during rewarming. However, at these low temperatures ventricular fibrillation is of a very fine nature, usually detectable only on the electrocardiogram. This provides a quiet heart in which to perform surgery whether there is cardiac arrest or ventricular fibrillation. The three dogs which did not fibrillate remained in cardiac arrest during the surgery and resumed a normal beat during rewarming. All other dogs were defibrillated easily with one or two shocks from a standard defibrillator. No relationship could be found between ventricular fibrillation and the rate of cooling or rewarming, the blood flow rate or the gas administered during the procedure.

Twenty-two of the animals were cooled at a rate of 1 degree per minute to temperatures of 13° to 18°C. and subjected to circulatory arrest for twenty-five to thirty-five minutes. During this time their temperature drifted upward 2

TABLE II
OXYGEN CONSUMPTION OF TEN DOGS DURING DEEP HYPOTHERMIA

Temperature (°C.)	O_2 Consumption (% normal)	Time Limits for Circulatory Arrest
37°C.	100	...
28°C.	50	8 to 10 min.
22°C.	25	16 to 20 min.
16°C.	12	32 to 40 min.
10°C.	6	64 to 80 min.

to 3°C. This upward drift during circulatory arrest is a constant observation with this cooling technic. The esophageal temperature represents that of the "core" organs such as heart, brain, liver, and the like. The muscle mass and subcutaneous tissues lag behind and are never cooled to the same degree as the internal organs. During circulatory arrest the internal temperature rises because of this differential. This observation has been made by Drew [1], Shields [3] and Kenyon [5] and in other studies by our group [6].

The eight animals which were cooled to the range of 8° to 12°C. had a slightly slower rate of cooling. This occurs because in the lower ranges the temperature differential between ice water and blood and between blood and body is much less. These animals had periods of circulatory arrest up to one hour, with an upward temperature drift of 5 to 7°C.

Rewarming always required a longer period of time than cooling. This is probably due to the following two factors: (1) during the initial stage of rewarming, blood flow is slower than during cooling; and (2) since the maximum water temperature used is 40°C., during the final stages of rewarming the temperature differential between blood and body is small.

Oxygen Consumption. In another group of ten dogs [7], oxygen consumption was studied at various temperatures. Similar studies have been performed by Bigelow [8], Gollan [9], and others. However, their studies did not extend into the temperature ranges in which we are interested for prolonged deep hypothermia. Accordingly, we have studied oxygen consumption at temperatures down to 10°C. in dogs whose temperature level and circulation have been maintained by extracorporeal pumps and a heat exchanger. Table II provides a brief summary of those results and a key to the acceptable time limits for circulatory arrest at various temperatures. In actual studies on dogs and in clinical experiences with patients, it has been found that no electroencephalographic, neurologic or other untoward effects have resulted when the circulatory arrest time has not exceeded the amount of time noted in this table. However, it is important to remember that this temperature represents the mean temperature during the total period of circulatory arrest, so that the amount of upward drift during this time must be anticipated.

TABLE III*
MEAN ARTERIAL PH VALUES IN FIFTEEN DOGS

No. of Dogs	Gas Breathed During Cooling and Rewarming	Arterial pH	
		Before Cooling	After Rewarming
5	100% O_2	7.37	7.28
5	98% O_2 2% CO_2	7.36	7.29
5	95% O_2 5% CO_2	7.33	7.27
15	Mean	7.35	7.28

* Depth of hypothermia is 9° to 15°C. Time of circulatory arrest is 25 to 60 minutes.

Studies of pH. The aforementioned studies of oxygen consumption indicate the level of over-all metabolism continuing at very low temperatures. This is further emphasized when rewarming is started after the period of circulatory arrest has approached the time limit. In such instances the initial blood coming from the right atrium is very dark and may have oxygen saturations in the range of 30 to 40 per cent. Acid metabolites are accumulating in the tissues during this period of circulatory arrest, and there should be a metabolic acidosis during the stage of rewarming.

Measurements of arterial pH were carried out on fifteen of the dogs prior to cooling, during cooling and at the completion of rewarming. These are summarized in Table III. The mean arterial pH was normal when cooling started and showed only a mild depression following rewarming. However, this degree of acidosis did not appear to be clinically significant in these animals and it was not neces-

sary to administer bicarbonate to expedite their recovery. These results are at variance with those of Kenyon et al. [5] in their experimental work on deep hypothermia in dogs. This group used an oxygenator in conjunction with deep hypothermia and found that survival of animals required the administration of a sodium bicarbonate–calcium gluconate solution. This has not been our experience in dogs or patients.

The pH values obtained at the deep levels of hypothermia are moderately and sometimes severely acidotic. However, these were measured at 38°c. and were not corrected for temperature. Furthermore, it is not known what physicochemical effects deep hypothermia has on the enzyme and buffer systems. Further studies in this area should be useful, but on a clinical level it is important to note that significant acidosis did not result following rewarming from deep hypothermia.

Hemolysis. Hemolysis has always been an important consideration in heart–lung bypass. The various types of mechanical oxygenators are a major source in the production of hemolysis. By eliminating the extracorporeal oxygenator and much of the accessory paraphernalia, hemolysis has become negligible with this system of deep hypothermia. It is usually below 25 to 50 mg. per cent and has not exceeded 75 mg. per cent even when the cooling and rewarming has required the pumps to be run for periods of sixty to eighty minutes. The before and after serum specimens for hemolysis usually show so little difference that we have stopped performing this determination in laboratory and clinical cases.

Blood Flow and Blood Pressure. The level of the reservoirs is adjusted in each case to provide the maximum outflow from the atria. This is usually achieved by having them fourteen to eighteen inches below the atrial level. Flow ranged from 40 to 90 cc. per kg. per minute with a mean of 60 cc. per kg. per minute. Higher flows could be achieved by increasing the animals' blood volume prior to starting the cooling. However, at flow rates of 90 to 140 cc. per kg. per minute, the rate of cooling and rewarming were not significantly increased. The blood pressure at low temperatures was higher when the higher flow rates were used, but this did not appear to be essential. With flow rates of 60 cc. per kg. per minute, the mean arterial blood pressure at deep hypothermia levels ranged between 40 and 80 mm. Hg prior to circulatory arrest.

TABLE IV
TWENTY-ONE SURGICAL PATIENTS OPERATED ON UNDER DEEP HYPOTHERMIA

Diagnosis	No. of Cases		
	Performed	Recovered	Died
Atrial septal defect			
Ostium secundum	2	2	...
With partial transposed veins	1	1	...
Total anomalous pulmonary veins with atrial septal defect	1	1	...
Aortic stenosis			
Congenital			
Valvular	2	1	1*
Supravalvular	1	1	...
Acquired (calcific)	1	1	...
Pulmonary stenosis			
Valvular	1	1	...
Infundibular	1	1	...
Combined valvular and infundibular with atrial septal defect	2	2	...
Ventricular septal defect			
Ventricular septal defect only	2	1	1†
Ventricular septal defect with pulmonary valvular stenosis	1	1	...
Tetralogy of Fallot complex			
Acyanotic	2	2	...
Cyanotic			
Tetrad only	1	1	...
Tetrad plus absent pulmonary valve	1	...	1
Tetrad plus atrial septal defect	1	...	1
Tetrad plus endocardial cushion defect	1	...	1
Total	21	16	5

* Technical error, the right coronary artery occluded by repair of aorta.
† Aspirated food on third postoperative day.

SURGICAL CASES

The same basic technic and equipment described have been used on twenty-one consecutive, unselected patients for the repair of a wide variety of surgically correctible cardiac lesions. Most of the usual operable lesions and a number of less common ones are included in this series. The general information on these patients is summarized in Tables IV and V. This reveals a broad spectrum of patients of various ages and weights and with different types of lesions in whom surgery has been satisfactorily performed under deep hypothermia.

There were five deaths in the series, giving an over-all mortality of 23.8 per cent. If the four cyanotic tetralogy complexes are excluded (three patients had additional and unusual lesions), the mortality rate for the acyanotic group is 11.8 per cent.

The temperatures used were specifically

TABLE V
TWENTY-ONE SURGICAL PATIENTS OPERATED ON UNDER DEEP HYPOTHERMIA

Data	Results
Age	3½ to 43 yr.
Weight	29 to 160 lbs.
Cooling	
Coolest temperature	8° to 21°C.
Rate of cooling	1½°C. per min.
Circulatory arrest	
Duration of arrest	18 to 60 min.
Temperature drift up during arrest	1° to 2°C. in 15 min. 3° to 4°C. in 30 min. 5° to 6°C. in 60 min.
Rewarming rate	¾°C. per min.
Incidence of ventricular fibrillation	All except two patients
Arterial pH	
Before cooling	7.36
After rewarming	7.35

selected for each patient on the basis of the time usually required for the repair of such lesions. In no case did the circulatory arrest time exceed the limits delineated in Table II. The longest period of circulatory arrest was sixty minutes. This patient's temperature drifted from 9° to 15°C. during this time. With his mean temperature of 12°C. his anticipated limit of circulatory arrest was fifty-three to seventy minutes.

Electroencephalograms taken before and after surgery were normal in all patients. All patients except one regained consciousness, were lucid and had normal reflexes and responses. The patient who did not recover from the surgery was a four year old Mongol with a tetralogy whose ventricular septal defect represented a variation of an endocardial cushion defect. The four patients who succumbed after surgery had normal sensorium and reflexes until their death. One of these aspirated food on the third postoperative day and another died twelve hours after surgery with a massive myocardial infarct resulting from a technical error, the right coronary artery orifice being completely obstructed by the closure of the aorta.

All except two of the patients had ventricular fibrillation during the cooling or rewarming. The hearts were effectively defibrillated at 28°C. or above by one or two shocks with a setting of 110 volts at 0.5 seconds, except for the patient with aortic stenosis in whom the right coronary artery was completely occluded by the repair of the aortic wall. Despite his massive infarct, the presence of which was not appreciated until postmortem examination, his heart was effectively defibrillated with multiple shocks and he lived for twelve hours.

There were no serious aberrations of acid-base balance and it was not necessary to give any patient bicarbonate or lactate solution postoperatively. The arterial pH's were near normal at the completion of rewarming but usually drifted downward during the first several hours postoperatively. However, these patients did not appear clinically to be acidotic and spontaneously corrected their pH by the following day.

The patients cooled faster than the dogs probably because the operating rooms were air conditioned and the internal cooling was supplemented by placing them on a mattress through which ice-water was recirculated. The cooling mattress was started at the same time as internal cooling. It was also used with warm water during and after rewarming to prevent a downward temperature drift.

During circulatory arrest the temperature drift upward averaged 1° to 2°C. in fifteen minutes, 3° to 4°C. in thirty minutes and 5° to 6°C. in sixty minutes. Knowing these values and the anticipated time for a repair, it is possible to calculate the mean temperature desired and the lowest temperature required for a specific operation. If the intracardiac surgery requires longer than anticipated or the temperature drifts up more than expected, the heart can be closed with sutures or a clamp and the blood recirculated and recooled to provide additional time for completing the repair. In three of our patients with multiple defects, such reperfusion and recooling was employed between repair of the different lesions. The use of temperatures down to 8°C. and the addition of reperfusion and recooling when necessary makes it possible to extend the period of circulatory arrest up to several hours.

COMMENTS

Several groups, including those of Pierce [*10*] and Sealy, Brown and Young [*11,12*] have used deep hypothermia in conjunction with an oxygenator. In view of the findings noted here in experimental animals and patients this would appear to be unnecessary. Furthermore, it requires more equipment and more blood with all

of the dangers and disadvantages of both. There are few opportunities for mechanical failure in the simple equipment used for deep hypothermia without an oxygenator. The total priming volume required is less than three units of blood. This same amount of blood is required for all patients, small children or large adults.

To the surgeon, the use of this technic provides an ideal field. It is completely quiet from arrest or fine fibrillation without the deleterious effects of drugs or anoxia. The field is also completely bloodless. There are no machines running during the intracardiac procedure and there is no flow through an atrial or ventricular defect, no return from the coronary or bronchial systems or from an unsuspected ductus. At the completion of the procedure before the heart is closed all air is carefully replaced by blood. The aortic clamp, which is always placed across the ascending aorta before the heart is opened, is kept in place during the initial phase of rewarming until there is assurance that no residual air remains in the left side of the heart.

The 10 per cent mortality in dogs and 23 per cent mortality in unselected, consecutive surgical cardiac cases in our experience establishes deep hypothermia without an oxygenator as the simplest, safest, and most efficient technic for intracardiac surgery at the present time.

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Elective Hypothermia During Extracorporeal Circulation with a New Heat Exchanging Filming Oxygenator

INDICATIONS AND RESULTS

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THE combined use of hypothermia and extracorporeal circulation has been studied in the experimental laboratory for many years [1]. Recently it has found clinical application in open cardiac surgery, largely through the work of Sealy et al. [2] and Sealy and associates [3].

Our interest in hypothermia as an adjunct to extracorporeal circulation began in December 1958, and since that time we have performed 172 consecutive perfusions incorporating some degree of hypothermia.

The first eighty-eight patients in this series were perfused with the aid of an oxygenator which did not provide for rapid heat exchange, and as a consequence the temperature of the blood was lowered only mildly between 30 and 34°C. This early experience was favorable in that the addition of hypothermia to cardiopulmonary bypass was associated with a decline in postoperative morbidity. Patients so treated had fewer respiratory complications, a decrease in the incidence of febrile reactions in the first forty-eight hours and a more rapid recovery.

In August 1958, we obtained delivery of a new oxygenator incorporating adequate heat exchange for rapid cooling and warming. This oxygenator has been described elsewhere [5] but in summary is a disc oxygenator modified from the original Bjork [6]. The greatest efficiency with a disc oxygenator (ratio of exposed area wet with blood to area of disc immersed in blood) is achieved when a large disc turns in a shallow trough of blood, with oxygenation occurring only near the periphery of the disc. But at a given rotary speed, disc size in practice is limited by the downward gravitational force and the outward centrifugal force which may lead to splashing of blood, foaming and destruction of blood. We designed a large disc oxygenator which would operate at high rim velocity without foaming by reducing the space between disc and casing to less than $\frac{1}{50}$ inch with the result that a meniscus of blood continuously spans the gap between disc rim and wall. (Fig. 1.) This prevents splashing, adds the entire inside of the casing to the oxygenating surface, and allows the casing to act as an efficient heat exchanger. A water jacket is provided on the casing to govern its temperature, and a shallow groove is provided under the discs to allow transit of blood from one end of the apparatus to the other.

This design greatly increases the oxygenating capacity of the oxygenator while decreasing its priming volume. Satisfactory oxygenation and prolonged perfusion of an adult patient have been achieved with a priming volume of 4 units of blood for the entire pump oxygenator tubing system. Because of the large area of moving blood in contact with the inner wall of the

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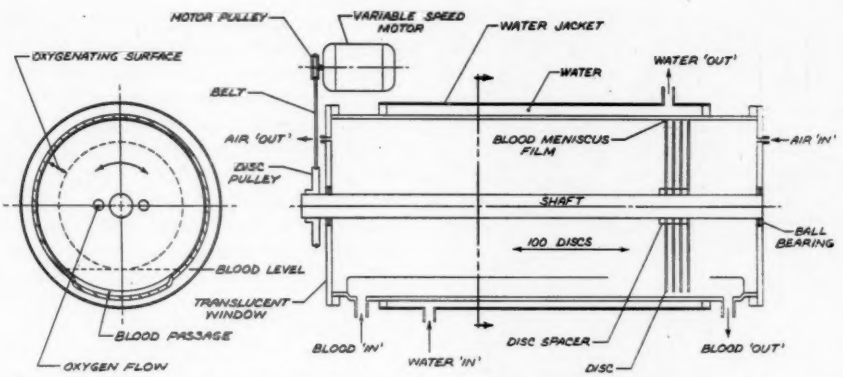

FIG. 1. Diagram of a cross section and longitudinal section of the oxygenator which incorporates a heat exchanger.

jacketed casing, heat transfer at a rate greater than 50 kg. calories per minute is possible. For example, a 280 pound man with aortic stenosis was cooled 10°C. in twenty minutes. A graph of the temperature change in a typical patient is presented in Figure 2.

This oxygenator has now been used in eighty-four procedures. As we became familiar with it, we began to cool patients to lower temperatures, mainly for the following three reasons.

First, it is sometimes difficult to maintain a normal "cardiac output" during perfusion at normal body temperatures even using "high flow" principles. Bottlenecks in the perfusion system can occur either at the arterial or venous ends, so that moderate to severe reductions in blood pressure can last for rather long periods. For instance a perfusion at a blood pressure of 80 mm. Hg is not uncommon, yet for many patients this is the pressure of mild shock. Such a necessary reduction in effective blood flow during perfusion is tolerated better in a hypothermic patient.

Second, and perhaps more important, we have evidence that the removal of blood from the operative field through the cardiac sucker accounts for almost all of the trauma to blood occurring during extracorporeal circulation. This aspirated blood, which is often heavily refluxed with air, may amount to over a liter a minute in cyanotic patients with large bronchial arterial flow. The realization that this was a serious complication suggested that any technic which would reduce bronchial flow yet permit safe perfusion would be advantageous. The reduced metabolism of deep hypothermia allows the safe use of lower rates of perfusion flow, and consequently less intracardiac suction damage to blood components.

Third, we discontinued using potassium cardioplegia because of the slowness of myocardial recovery and the frequency of arrythmias after perfusion. Undoubtedly, the concomitant obligatory cardiac anoxia accounted for many of these disadvantages. Our own experimental experience, and the report of Berne, Jones and Cross [4] suggested that profound hypothermia would be the method of choice for the treatment of complex intracardiac lesions when a quiet heart was desirable. Although we had investigated selective cardiac hypothermia in the laboratory it seemed more complex to manage than general body hypo-

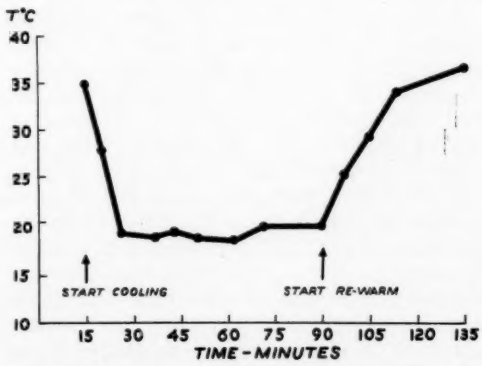

FIG. 2. A graph to show the relationship between temperatures and time during cooling and rewarming using the heart-lung machine in a representative patient.

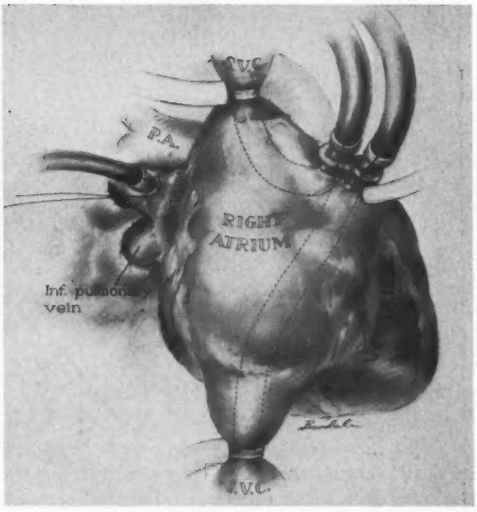

FIG. 3. Drawing showing the heart retracted to the left to expose the left atrium and the right pulmonary vein allowing insertion of cannula for drainage of left atrium.

thermia. We also found that cold fibrillation was as satisfactory as standstill from the operative point of view, and quite easy to manage.

The depth of hypothermia used has depended on the complexity of the anticipated intracardiac problem. Thus, atrial septal secundum defects and pulmonary stenosis were kept in the range of 30 to 34°C., the ventricular septal defects and endocardial cushion defects were cooled to between 20 and 30°C., while the cyanotic tetralogies were taken to the range of 16 to 26°C.

Ventricular fibrillation or cardiac standstill has not occurred at temperatures above 29°C. However, it is usual at lower temperatures, 70 per cent of our patients fibrillating at a mean temperature of 26°C.

We believe that left atrial decompression is an important feature of the operative management whenever cold fibrillation is caused or contemplated. Various places of access were tried, including the left atrial appendage and right transatrial, but the most satisfactory has been through the portion of the left atrium which presents posteriorly on the right. (Fig. 3.) After partial bypass is started, a perforated catheter is inserted through two purse string sutures into the left atrium in the insertion of the right superior pulmonary vein. The inner purse string is used to anchor the catheter while the outer one closes the stab wound when the catheter is removed at the end of the procedure. Drainage is provided into the coronary reservoir, from which there is also regulated suction. A side-arm on the tube permits injection of blood into the left side of the heart to remove residual air at the conclusion of the repair. Left atrial decompression has been used mainly during operation for large interventricular septal defects, tetralogy of Fallot, aortic valvular lesions, and whenever hypothermic fibrillation or standstill are contemplated.

With a heart-lung machine in the circuit, ventricular fibrillation during cooling may be an asset rather than a liability. Defibrillation is easily accomplished by a single electric shock after the patient has been rewarmed to 33 to 34°C., and has never been a problem. About 20 per cent of our patients have reverted spontaneously from ventricular fibrillation to sinus rhythm while warming, usually at a temperature near 32°C.

Approximately 30 per cent of the patients taken below 25°C. were found to have complete heart block during rewarming. This disappeared spontaneously prior to coming off perfusion in the majority of cases. The use of a pacemaker in the early postoperative period was required in a small number of patients so cooled. In every instance heart block has spontaneously disappeared within the first forty-eight hours following surgery, so that in our hands profound hypothermia has resulted in a decreased incidence of permanent heart block.

Few drugs are used. Calcium chloride or calcium gluconate is used to balance the citrate from banked blood. Prostigmine® is employed to break a resistant ventricular tachycardia and Isuprel® is used in the event of complete heart block. In general we do not favor the use of digitalis preparations during open heart surgery, and they are used with caution during the recovery period.

The addition of hypothermia in general has resulted in a lower postoperative morbidity, while the use of profound hypothermia (below 30°C.) has resulted in addition, in a lower mortality from surgery in those lesions which previously had proved the most hazardous to correct, for example, tetralogy of Fallot. Table 1 illustrates the last eight consecutive operations for cyanotic tetralogy of Fallot. Aortic cross-clamping has been found to be

TABLE 1
EIGHT CONSECUTIVE OPERATIONS FOR CYANOTIC TETRALOGY OF FALLOT PERFUSED WITH HYPOTHERMIA

Age of Patients (yr.)	Previous Surgery	Cyanosis	Clubbing	Hematocrit (%)	Perfusion Time	Lowest Temperature	Gusset in Outflow Tract	Result
9	Left Blalock	Marked	Moderate	68	1′ 43″	26.4°C.	Yes	Complete correction
9	Left Blalock	Marked	Marked	79	1′ 21″	25°C.	Yes	Complete correction
2	None	Marked	Marked	68	4′ 32″	23.5°C.	Yes, including bifurcation of pulmonary artery	Death in early part of operative period
6	Left Blalock	Marked	Marked	74	1′ 46″	23°C.	Yes	Complete correction
7	None	Mild	Moderate	48	1′ 32″	19.7°C.	No	Complete correction
6	None	Marked	Marked	80	2′ 29″	19°C.	Yes	Complete correction
7	None	Moderate	Mild	58	1′ 57″	16.5°C.	Yes, out to bifurcation of pulmonary artery	Complete correction
15	Left Blalock Right Blalock Pulmonary valvotomy	Mild	Moderate	50	2′ 18″	20°C.	No	Complete correction

necessary only when there has been aortic incompetence. Occasionally, short periods of aortic occlusion are desirable during the repair of a difficult tetralogy or large interventricular septal defect but in these instances we have not allowed the coronaries to remain unperfused for longer than ten minutes at a time. In the treatment of congenital and acquired aortic stenosis, when prolonged dissecting on the valve was not necessary we have used a combination of general hypothermia and extracorporeal circulation without coronary perfusion. Despite the known fact that a heart will recover from prolonged periods of cold cardioplegia without coronary perfusion we believe it best to employ it at least intermittently, through cannulas, when extensive procedures are necessary.

The work of Niazi and Lewis [8,9] clearly showed that biologic recovery is possible after fifty to sixty minutes of completely arrested circulation at temperatures below 10°C., and Drew [7] has utilized this information for the performance of open heart surgery without the use of an artificial oxygenator. Cooling and rewarming are performed by utilizing two pumps only, one to pump oxygenated blood from the left atrium to the femoral artery, and the other to pump caval blood through the lungs.

It has been our impression after studying this method experimentally that the cannulation is more cumbersome than when an artificial oxygenator is used, and that this would be most disadvantageous in the type of patients for whom profound hypothermia is most useful such as tetralogy of Fallot with a diminutive outflow tract. We have found it often is desirable to return to partial or total perfusion after a difficult repair because a test of the circulation has shown that the repair should be revised or additional work is necessary on a valve. Furthermore, some patients require partial perfusion for twenty to thirty minutes before satisfactory circulation is established. In such circumstances a complete pump oxygenator presents less difficulty than a double cannulation system.

SUMMARY

1. Hypothermia has been used in conjunction with extracorporeal circulation in 172 consecutive cases.

2. The last eighty-four procedures have been performed with a new disc oxygenator with an integral heat exchanger allowing for the election of more profound hypothermia.

3. Mild hypothermia has the effect of lowering the postoperative morbidity following cardiopulmonary bypass, whereas, profound hypothermia with extracorporeal circulation has resulted in a decreased mortality from the surgical correction of the most complex intracardiac lesions.

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DISCUSSION OF PAPERS BY DRS. GORDON, JONES, LUDDINGTON AND MEYER; AND GERBODE, OSBORN, BRAMSON, HARKINS, ROSS AND JOHNSTON

Frank Gerbode (San Francisco, Calif.): We took a long, hard look at the Drew technic when Dr. Drew described it in England. However, we decided that although there are advantages in using hypothermia in conjunction with some system for pumping blood in open heart surgery, incorporating an oxygenator in the system increases the safety and allows for considerable more versatility during difficult procedures.

We believe that some physicians using hypothermia in conjunction with pump oxygenators do so because they believe that their oxygenator is inadequate. If it is, one can use low flow with reduced body temperature and have safer perfusions. This is not the reason that we have adopted hypothermia, for our oxygenator has never been inadequate in meeting any situation under normothermic conditions.

There are a number of reasons why we need to have an oxygenator in the system in this type of work. First of all, we have noticed quite frequently that the cold heart during recovery needs support for some period of time. It is our impression that to continue a partial to full perfusion during this time with a complete pump oxygenator system is usually simple.

We also have a number of patients in whom it is necessary to revise some part of the repair. This happens more frequently in the tetralogy group, but has happened following the repair of other defects as well.

Another advantage of having an excellent heat exchange unit is that it allows for rapid cooling and rewarming.

Much has been said about the danger of fibrillation with hypothermia in these systems. However, fibrillation is really no problem. In fact, it usually is a welcome addition for it affords a quiet, slowly fibrillating tight heart to work on, easily as satisfactory as using potassium cardioplegia.

One must keep an open mind about this whole field for there are many unanswered questions.

John C. Jones (Los Angeles, Calif.): Dr. Gerbode and his associates have presented a matchless contribution in the field of open heart surgery. They have succeeded in if not perfecting, in almost perfecting, a combination of oxygenator and heat exchanger in one unit that they have proved is atraumatic to the blood elements. It is most efficient in its work at low flow rates, along with the economy of the blood volume that is required in priming the pump. The ease of cleansing and sterilizing the unit should be stressed. That in itself is a big factor because, in a busy clinic, it requires a good deal of help to do this work. If this can be simplified such as the unit can do, it will be a big contribution in itself.

I believe that these features have certainly fulfilled the cardiac surgeon's needs in almost all of the aspects of bypass technic, and particularly impressive are the low flows required by virtue of the low temperature plus an efficient oxygenator. These aspects make this technic most attractive to the surgeon.

There is a distinct advantage in eliminating the pulmonary artery catheter. It has to be carried in

through the stab wound in the ventricle to insure an adequate pulmonary outflow tract for oxygenation. But we, as yet, have not had any particular difficulties either in the atrial or ventricular defects that have been corrected with the pulmonary catheter in place during the use of the pump.

Briefly, in our monitoring of CO_2 alveolar air during anesthesia in cases of regular hypothermia and profound hypothermia, technic was worked out for us by Dr. Digby Leigh, the anesthesiologist at Children's Hospital. He employs a continuous monitoring of the alveolar air through a fine plastic catheter, which is continuously recorded through this rapid CO_2 infrared analyzer. We believe that it has been a great help to us. It has eliminated a lot of blood pCO_2 and pH determinations and guesswork as to just where we stand long before we go into the hypothermia phase of surgery, and obviously if we have a patient that has not done well before this, we already have two strikes against us. Our anesthetist is able to warn us that things are not going well immediately with this little analyzer.

In an ordinary hypothermia with occlusion of the vessels for pulmonary valvulotomy, as we clamp off the vessels, or course, the inflow and outflow tracts are completely occluded. The minute we come off with the circulation restored we have the record. This is only 2½ per cent CO_2 but that is just about where we run on the hypothermia. With ordinary temperatures, this would be around 5 or 5.4 per cent. So that we know, immediately after the outflow and inflow tracts are opened, that there is a good heart output and the circulation to the lung is excellent by virtue of the heart's action.

While performing lobectomy for a tension cyst in an infant, the anesthesiologist told me that I was in trouble. Obviously I was placing too much traction on the hilum, causing complete bronchial occlusion. Immediately after the traction was removed there was a good CO_2 output and normal aeration of lung.

In a case of a cardiac arrest, the resident was trying to carry out cardiac massage while the atrium was being sutured, or some repair work was being performed. Obviously this cardiac massage was not efficient. Immediately, however, on establishing efficient cardiac massage the CO_2 output was good and the situation under control even though the massage went on.

In some patients, particularly those in whom the surgeon is preparing to introduce catheters through the left atrial appendage, there is a good deal of manipulation of the heart and disturbance of the inflow and outflow tracts which results in a CO_2 output at a dangerously low level. On alleviating that situation it returns to a normal level.

Benson B. Roe (San Francisco, Calif.): I am delighted to see the resurgence of hypothermia. We became familiar with it because we were performing open heart surgery with inflow occlusion and hypothermia before oxygenators were successfully introduced in this particular area. We were impressed with the advantages, safety, simplicity and low morbidity that have been emphasized. The use of hypothermia in conjunction with the extracorporeal circulation is, I think, the adequate answer to many of our problems.

We still use hypothermia with inflow occlusion without the use of the oxygenator for simple lesions, and have the oxygenator standing by in case the lesion turns out to be more complicated than we anticipated. This procedure is not because of any dissatisfaction with our oxygenator, but rather because of the virtues which have been emphasized previously.

I caution Dr. Gordon's group, however, because although the metabolic curves are significantly shifted to the right in the presence of hypothermia at low levels, there is no question that significant ischemic effects occur that are only partially reversible. The fact that we can get away with it in a large majority of patients obscures the hazard. The metabolites do pile up and the oxygen debt has significant disadvantage, so that when any significant period of work is anticipated, one would favor a technic that would perfuse both the heart and brain.

We do not arrest the circulation totally, except when a previous surgical shunt has been performed which cannot be exposed and closed.

I would agree that the ischemic types of arrest are to be abandoned because of their hazards which have been documented in a number of cases. We no longer use potassium citrate arrest for this reason.

Certainly the principal trauma to blood does occur from the cardiotomy suction, and we have fortunately answered that question by using a volume displacement type of suction instead of high vacuum, which is run as slowly as possible to avoid sucking air.

However, in using hypothermia for a cardiac arrest, when this becomes technically necessary, we have found some disadvantage in the fact that the heart arrests in spasm which sometimes interferes with adequate retraction and exposure.

I would also agree that defribillation is never a problem when adequate perfusion has continued.

I submit for your consideration our technic for combined hypothermia. All of our patients have had total body hypothermia to temperatures between 33° and 28°C., partially by accident in the early series because we had a metal reservoir which came into relative equilibrium with the room temperature.

We have recently added a system which combines simplicity and versatility without the necessity of an additional pump for coronary circulation. By this method we can cool the patient using the heat exchanger and blocking off the bypass line, so that

the perfusion is cooled or warmed as desired. We do not use cardiac arrest with hypothermia unless the situation demands it. We are rigged for coronary perfusion and can unclamp the line which bypasses the heat exchanger to divert the major flow to the femoral artery. The minor flow from the "Y" tube then passes through the heat exchanger (being recirculated from an ice tank) to the coronary circulation through the clamped ascending aorta. In this way the heart can be cooled rapidly, arrested rapidly, and rewarmed merely by removing the aortic clamp.

The saddle perfusion needle is secured with a tape tied in this groove. The point is thus maintained in aorta where it will not traumatize the aortic valve or the posterior wall.

These authors have contributed importantly to the progress in this field. The problems are not all solved but the combination of perfusion and hypothermia is an important step.

LYMAN A. BREWER, III (Los Angeles, Calif.): Certainly the work of Dr. John Jones' group has many advantages from the point of view of simplicity that commend it, and I hope they will continue it so that we will know their long term results.

There is one question regarding this technic. Do they have any difficulty in cannulating the pulmonary artery having a small outflow? Often one of the major reasons for surgery is the enlarging of the ventricular outflow tract and the pulmonary artery, perhaps even putting a patch on the pulmonary artery to give a sufficient diameter.

We have used hypothermia in conjunction with our pump oxygenator in a way somewhat similar to that which Dr. Roe previously presented. We went from the Melrose technic to anoxic arrest, and not being too satisfied with those technics, have gone on to the Brown-Seeley type of heat exchanger to produce cold arrest of the heart during surgery.

Attached to the oxygenator is a second outflow track which is set up so as to be always available. The second outflow track passes through the heat exchanger. An occlusive pump of the DeBakey type forces the cold blood to the clamped aorta, so that the coronary arteries are perfused with cold blood. The "femoral" line sends normothermic blood to the femoral artery.

In our experience by cooling the water to 4 or 5°C. in the heat exchanger, the blood will cool to 11 degrees and in one to three minutes cardiac arrest occurs. In comparing the Melrose and anoxic technics with the cold technic, the heart is entirely different. The heart is pink and a little firmer than normal, but it certainly seems to be in a much better physiologic state than with the anoxic or chemical methods.

The problem of defibrillation has not bothered us. We have started these hearts very easily and, as Dr. Gerbode pointed out, the period of defibrillation when you are on perfusion or returning to perfusion, may not be a problem but an asset.

We have studied these patients, too, from the point of view of the amount of cardiac trauma produced by the various technics. For example, if you study the glutaminic transaminase levels of the blood after operation, you have some interesting findings.

First of all, we have shown that in open thoracotomy and in closed cardiac technics, such as those for mitral stenosis, there is slight rise of the serum glutaminic transaminase level following surgery. We found with our disc pump oxygenator (a modified Kay-Cross type), there is no difference between an atriotomy on a pump oxygenator and a simple closed mitral atriotomy. Along with other physiologic studies, the pH of the blood and CO_2, we believe that the oxygenator as we are using it is a physiologic oxygenator.

We have been interested in cases in which an incision has been made in the ventricular wall, where rather large rises in the transaminase level are found as compared with atriotomy.

We believe that it is also a help prognostically, because if the transaminase level is high and remains high during the first twelve to forty-eight hours after surgery, there will be trouble as far as that heart is concerned. Therefore, this is a test that gives one an index of surgical cardiac damage. We had hoped that our cases in which this cold arrest was used would show significant changes in the transaminase level. They have not as yet, but we have not explored this enough to really know.

The combination of hypothermia and an oxygenator has certain advantages. Therefore, I believe the oxygenator described by Dr. Gerbode may result in an improved technic. At the present stage of open heart surgery, a number of years have been spent in the laboratory getting our programs going. When one has a program that is going well, he is loath to change in a hurry. Therefore, I hope this work will continue and gradually, out of this rapidly changing picture, we will, I am sure, evolve simpler and more practical technics.

ARCHER GORDON (closing): I think what Dr. Brewer has stated about each one developing his own technic and being somewhat loath to change is quite true.

Regarding the use of deep hypothermia without an oxygenator, I would say that having worked with an oxygenator and with an oxygenator plus hypothermia, it was a real revelation to us to use deep hypothermia alone. It is hard to explain unless you have done it or seen it.

With deep hypothermia alone the heart is absolutely quiet without drugs and without anoxia. The heart is absolutely bloodless. When the proper temperature is reached, the tapes are pulled up on

the venae cavae and the heart is emptied of blood. There is no machine running, no bronchial return, no coronary return and no blood rushing back into the pulmonary artery from a ductus which has not been detected previously. This is an ideal field for surgery to be performed. I believe that once others have seen or worked in such a field, they will realize how ideal it is.

This alone does not justify the technic unless we can do it without jeopardizing the patient. In the experiences we have had, the results speak for themselves. The patients have not been jeopardized. However, I feel sure that if this technic is not used carefully, operators may exceed the limits of circulatory arrest, and deleterious effects of prolonged arrest are going to be seen. This is not an indictment of the technic but rather an indictment of people who might use this technic improperly. We have found it very beneficial and hope to continue using it.

One advantage I failed to mention in addition to simplicity of equipment is the small amount of blood used. The amount of blood is less than 3 pints for the entire set-up. This applies to cases involving children or adults. The priming volume is the same for all patients. This can probably be reduced further when we improve the physical set-up of our equipment.

The catheter in the right ventricle, mentioned by Dr. Gerbode, has not been a problem. The catheter is small enough so that flow around it is quite adequate. There does not seem to be regurgitation through the pulmonary valve; at least not enough to produce any significant effect.

In our routine, as we approach the end of the rewarming period the level of the gravity flow reservoirs is raised, so that the patient's heart gradually takes over the function of propelling the blood through the lung and the body. After the level has been raised and the heart has resumed its pumping function gradually, we then turn off the pumps. There has not been a problem with obstruction of the outflow tract by the catheter.

We have not yet had an outflow tract or a pulmonary artery which was so small that it created a special problem. In cases of valvular pulmonary stenosis or significant infundibular stenosis, the catheter has been placed in the pulmonary artery rather than in the outflow tract of the right ventricle. These cases usually have a poststenotic dilatation and placing the catheter in the pulmonary artery has not created a problem.

I am sure that in time better and better oxygenators will be made. Right now we believe that we have the best oxygenator, namely the human lung, and hope that subsequently we can again compare notes and see which of these technics has turned out to be the best for all.

GEORGE A. HARKINS (closing): I would prefer to emphasize the similarities that we find in Drs. Gordon, Jones and Meyer's work rather than emphasize the differences.

I think Dr. Gordon has described what we find to be an excellent operating situation, which is a quiet heart with little activity needed around it. As a matter of fact, when we get down into the depths of profound hypothermia with our oxygenator, Dr. Osborn has occasionally asked Dr. Gerbode if he would like to have the circulation turned off so as to cut out the noise also.

We have been doing physiologic studies during the course of hypothermia with extracorporeal circulation. We find an interesting thing happens to the CO_2 when we cool these patients rapidly. It would seem that during the course of cooling, the body rapidly absorbs CO_2. This begins to drop the blood pH, but we find that if more CO_2 is given and further respiratory acidosis induced the perfusion seems to go better and the postoperative course seems to be smoother. If we prebreathe with CO_2, fibrillation spontaneously reverts on rewarming in more than 25 per cent of patients, which suggests that the body wants CO_2 during cooling. During rewarming there is an almost explosive release of CO_2 from the tissues, and we certainly recommend an adequate oxygenator to cope with that. I am certain that Dr. Jones' oxygenator, the human lung, and ours can.

In response to what Dr. Roe stated in regard to hypothermia, we consider hypothermia without extracorporeal circulation to be an entirely different situation, and although it is handy and simple in some cases, it does not, in our opinion, give the versatility or, more important, the safety that you get with hypothermia in combination with extracorporeal circulation.

Indications for Measurement of Arterial Blood pH

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ALMOST all of the functions of the body depend upon chemical reactions which demand a relatively constant hydrogen ion concentration or pH. To preserve the integrity of these reactions, the pH of arterial blood is normally maintained within the very narrow limits of pH 7.40 to 7.43. Minor variations of pH can provoke physiologic dysfunctions that may add considerably to morbidity. Major deviations of pH, are incompatible with life unless they are promptly corrected. Alterations of pH, commonly known as acid-base disturbances, are frequently encountered among surgical patients. Therefore, it is important that the surgeon have laboratory tools available which will provide a correct diagnosis in all instances of acid-base imbalance. Although it has been known for many years that the level of CO_2 in the blood does not provide definitive information as to the hydrogen ion concentration [1], the majority of clinicians still rely principally on alterations in blood or plasma CO_2, or CO_2 combining power, for the detection and evaluation of disturbed acid-base equilibrium [2].

In the past six years, our experience with a simple, reliable glass electrode pH meter has demonstrated that the direct measurement of the arterial blood pH is essential in the proper evaluation and management of acid-base disorders. Dependence on measurement of blood CO_2 alone may lead to serious errors in diagnosis. Moreover, an important number of life threatening hydrogen ion imbalances have been encountered that could *only* have been detected and measured by the arterial blood pH because in these instances the blood CO_2 value was normal.

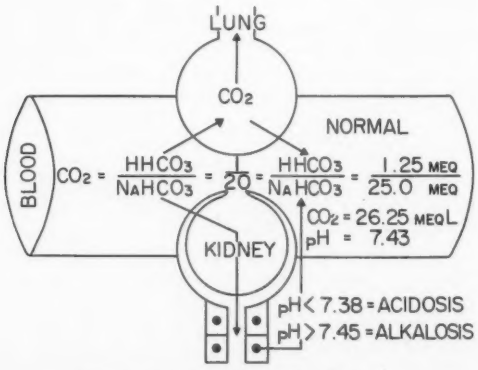

FIG. 1. Regulation of blood pH.

REGULATION OF ARTERIAL BLOOD PH

It is important to recognize that blood CO_2 is not a measure of either acidity or alkalinity. The blood or plasma carbon dioxide consists of two components: carbonic acid ($HHCO_3$) and bicarbonate ($NaHCO_3$). Together, these form the bicarbonate buffer system and consitute a major element in the regulation of the pH of the blood. Normally carbonic acid ($HHCO_3$) and bicarbonate ($NaHCO_3$) exist in a balance of 1 part $HHCO_3$ to 20 parts $NaHCO_3$. Therefore, if the blood CO_2 content in a normal individual equals 26.25 mEq./L., this represents 1.25 mEq. $HHCO_3$ and 25.0 mEq. $NaHCO_3$. (Fig. 1.) The ratio of $\frac{1}{20}$ ($HHCO_3$ to $NaHCO_3$) is important since it is by preserving this balance that the body maintains a normal blood pH. No matter what alterations occur in total CO_2 the pH will be normal if this ratio is retained. If, however, the concentration of

carbonic acid ($HHCO_3$) exceeds $\frac{1}{20}$ $NaHCO_3$ (either because of increased $HHCO_3$ or decreased $NaHCO_3$) the pH falls, or if there is a relative excess of bicarbonate ($NaHCO_3$) (either due to decreased $HHCO_3$ or increased $NaHCO_3$) above that in the ratio $\frac{1}{20}$ the pH rises.

In health, either the $HHCO_3$ or $NaHCO_3$ of the blood can be adjusted by respiratory and renal mechanisms. The $HHCO_3$ of arterial blood is in constant equilibrium with the CO_2 in the alveolus of the lung. Any variation in ventilation that raises or lowers the concentration of alveolar CO_2 is reflected immediately by an increase or decrease in arterial blood $HHCO_3$. Since this unbalances the bicarbonate buffer ratio, the pH promptly falls or rises. Any primary change in pulmonary ventilation produces an obligatory alteration in the arterial blood pH. In addition, an adaptive ventilatory change can restore the pH towards normal. $NaHCO_3$, on the other hand, filtered by the glomerulus of the kidney, can be reabsorbed in variable amounts and returned to the blood. Therefore, the regulation of the blood bicarbonate level is largely a function of the distal renal tubule. This renal mechanism functions more slowly than the pulmonary. The adaptive increase or decrease of bicarbonate reabsorption, however, can also restore the buffer balance and return the pH towards normal [3]. In health, the lung and kidney are coordinated to maintain an almost constant ratio of $HHCO_3$ and $NaHCO_3$ at $\frac{1}{20}$. The blood or plasma CO_2 remains approximately 25 to 27 mEq./L. and the pH stays between 7.40 and 7.43.

With disturbed ionic equilibrium, any pH less than 7.38 indicates significant acidosis and a pH above 7.45 indicates significant alkalosis. An acidosis with a pH below 7.30 or alkalosis with a pH above 7.50 represent very serious imbalances and a pH approaching 7.0 or 7.80 is incompatible with life unless corrected immediately.

ABNORMALITIES OF BLOOD PH

Either respiratory or metabolic disturbances can cause an elevation or depression of the blood CO_2. In the former, this is due to a primary alteration in the $HHCO_3$, and it occurs in the latter because of a primary change in bicarbonate. Since the effect on the hydrogen ion concentration and the therapy indicated are quite different, it is important to differentiate these pathologic states. The blood CO_2 measurement does not indicate the relative values for $HHCO_3$ and $NaHCO_3$. However, with the blood CO_2 and the arterial blood pH both metabolic and respiratory acidosis and alkalosis can be readily identified.

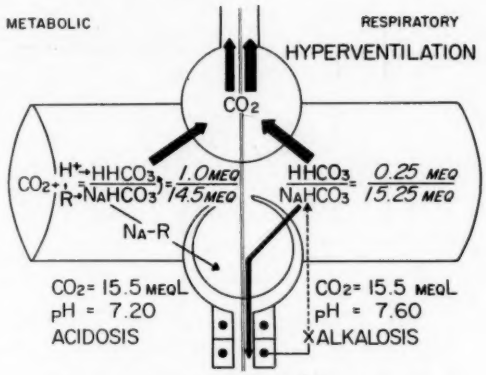

FIG. 2. Decreased blood CO_2 (15.5 mEq./L.).

Low CO_2. For example, the laboratory may report a low blood CO_2 (e.g., 15.5 mEq./L.) (Fig. 2.) If metabolic in origin, this can be due to an accumulation of excess organic acids in the blood as in diabetes mellitus, shock, or acute or chronic renal insufficiency. The organic acid radical combines with sodium and is excreted if renal function is adequate. The bicarbonate which has been displaced from sodium in the serum, combines with the excess hydrogen ion, forms carbonic acid, is liberated as CO_2 into the lungs ($HHCO_3 \leftrightarrows H_2O + CO_2$) and is exhaled by an adaptive increase in alveolar ventilation. The net effect is a decrease in the blood CO_2 but there is a relatively greater decrease in bicarbonate than in carbonic acid. Since there is then relatively more $HHCO_3$ than in a ratio of $\frac{1}{20}$, the pH falls. This is a metabolic acidosis and is identified by the low pH and low CO_2.

The same degree of lowering of blood CO_2 but with an elevation of pH will be produced by hyperventilation. (Fig. 2.) The latter may be precipitated by such disorders as hypoxia, hyperthermia, burns, thyrotoxicosis, diseases of the liver, psychic disturbance, or organic brain disease. With hyperventilation, excessive elimination of CO_2 in the lungs lowers the blood $HHCO_3$. In response, renal reabsorption of bicarbonate decreases. There is again a low blood CO_2 but since there is a greater loss of $HHCO_3$ and consequently relatively more $NaHCO_3$ than in the ratio $\frac{1}{20}$, the pH rises.

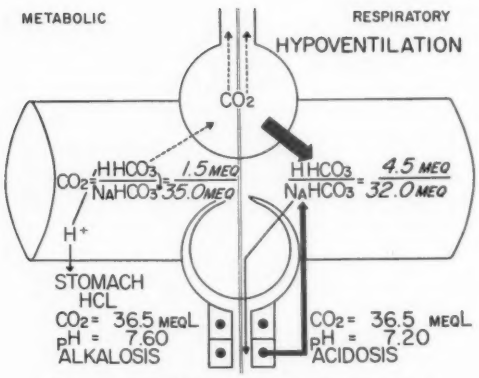

FIG. 3. Increased blood CO_2 (36.5 mEq./L.).

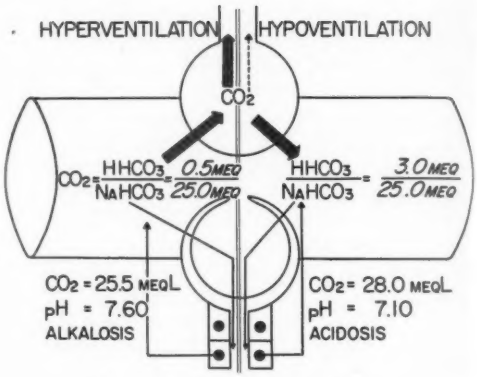

FIG. 4. Acute respiratory change.

This is identified as a respiratory alkalosis by the high pH and low CO_2.

In both of the foregoing situations, the level of lowered blood CO_2 is identical (15.5 mEq./L.). In one instance this is due to metabolic acidosis and in the other it is due to respiratory alkalosis. Laboratory differentiation without a blood pH measurement is impossible. When the arterial blood pH is known, however, precise differentiation becomes obvious.

High CO_2. When an elevated blood CO_2 is reported by the laboratory (e.g., 36.5 mEq./L.), a common metabolic setting is that of pyloric obstruction with active gastric secretion. (Fig. 3.) Excessive hydrogen ion and chloride are lost from the stomach as HCl. Excess serum sodium then combines with the excess HCO_3 elevating the $NaHCO_3$ component of the blood [$NaCl + HHCO_3 \rightarrow HCl$ (gastric secretion) + $NaHCO_3$ (blood)]. Through adaptive hypoventilation, retention of CO_2 is accomplished which augments the blood $HHCO_3$. The blood CO_2 content is elevated. Since there is a relatively greater increase in $NaHCO_3$ than in the ratio $\frac{1}{20}$, the pH rises and alkalosis exists. Another very common but frequently unrecognized mechanism also may elevate the blood CO_2, namely, hypoventilation due to narcosis, or occurring during anesthesia or postoperatively in patients with pneumonia or atelectasis or retained bronchial secretions. Here, decreased elimination of CO_2 in the lungs leads to an accumulation of excess $HHCO_3$ in the blood. (Fig. 3.) Adaptive renal reabsorption of bicarbonate augments the $NaHCO_3$ component of the blood but since there is relatively more carbonic acid than in the ratio (1:20), the pH falls. With the decreased arterial blood pH this disturbance is identified as a respiratory acidosis with a high CO_2 and a low pH. Only direct measurement of the blood pH can definitely differentiate these identical elevations of CO_2 as metabolic alkalosis in one instance and respiratory acidosis in the other.

Normal CO_2. It is of great importance also to note that changes in ventilation can produce dangerous degrees of alteration in the blood pH very quickly. In these instances only the $HHCO_3$ component of the blood changes and since this fraction is very small to begin with (1 to 2 mEq./L.), there may be no apparent change in the CO_2 content of the blood. For example, hyperventilation can reduce the $HHCO_3$ by 50 per cent in a very few minutes with a marked increase in pH. (Fig. 4.) Likewise, hypoventilation, even during relatively short intervals of anesthesia, can raise the $HHCO_3$ to two or three times normal and depress the pH into a life threatening range [4] even though the CO_2 combining power measurement is within the normal range. We have observed this phenomenon quite frequently and routinely measure the arterial blood pH in all patients who do not recover properly from their anesthesia or who have respiratory depression or hypotension the cause of which is not apparent. Many of these patients are also cyanotic and are commonly treated with ordinary oxygen therapy. Such therapy further reduces ventilation and aggravates the acidosis which may prove fatal [5,6]. All too often such deaths are erroneously attributed to hypoxia or some occult mechanism [7]. The prompt recognition of an acute respiratory acidosis by blood

TABLE 1

pH	Diagnosis	CO_2	Cause
Below 7.38	Acidosis	High	Respiratory
		Normal	Acute respiratory or mixed
		Low	Metabolic
Above 7.45	Alkalosis	High	Metabolic
		Normal	Acute respiratory
		Low	Respiratory

pH measurement in a comatose or hypotensive patient and proper therapy with oxygen using intermittent positive pressure breathing equipment [8], and intravenous administration of bicarbonate solution has been life-saving on many occasions (Case 1).

Finally, mixtures of metabolic and respiratory abnormalities at times combine to produce additive derangements of pH which likewise alter the CO_2 content of the blood very little. These also can be recognized only by pH determinations. For example, a patient with mild diabetes and mild pulmonary insufficiency postoperatively may have the diabetes go out of control. An excess of organic acid is thereby added to the blood and combines with sodium, displacing HCO_3 which should then be excreted through the lung by increased ventilation. Unfortunately, this patient is incapable of augmenting his ventilation because of the pulmonary disease. Also, he may already be hypoventilating because of the postoperative state. Consequently, the alveolar CO_2 is increased as is the blood $HHCO_3$. Renal bicarbonate reabsorption may already be maximal and cannot increase. Thus, the actual CO_2 content of the blood may not deviate from normal but the ratio is altered in that there is more $HHCO_3$ and less $NaHCO_3$. The result is a mixed metabolic and respiratory acidosis which may be extreme [9]. In the example (Fig. 5), this is quickly identified by the very low pH (7.10), but it would be impossible to detect it by measurement of blood CO_2 alone since the value obtained would be normal.

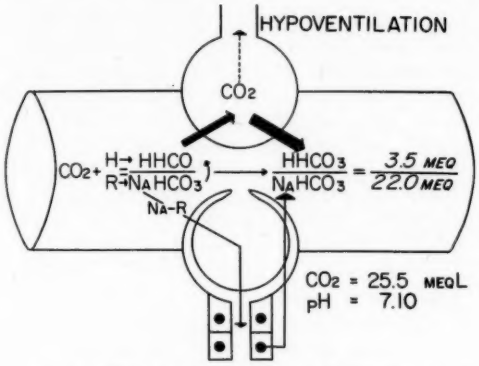

FIG. 5. Mixed metabolic and respiratory acidosis.

APPLICATION OF pH DETERMINATIONS IN EVALUATING ACID-BASE DISORDERS

From the foregoing discussion, it becomes apparent that when the arterial blood pH is measured and variations of blood CO_2 are evaluated in relation to the pH, acid-base derangements are easily and definitely categorized, both as to degree and cause. Proper therapy then becomes obvious and can be administered vigorously without any hesitation or doubt. Thus, *if it is known that the pH is low, acidosis exists.* (Table 1.) A low CO_2 identifies the acidosis as metabolic in origin and a high CO_2 demonstrates that it is due to pulmonary insufficiency. A normal CO_2 indicates either a respiratory acidosis of such short duration that renal adaptation has not yet occurred or that the patient has a mixture of metabolic acidosis and pulmonary insufficiency and requires therapy for both. *When the pH is known to be high, alkalosis exists.* A high CO_2 then indicates a metabolic origin and a low CO_2 demonstrates that the imbalance is due to hyperventilation. Combined metabolic and respiratory alkalosis can also exist with a normal CO_2 and high pH but such conditions are rare. These findings (i.e., high pH and normal CO_2) usually indicate a respiratory alkalosis in which there has not yet been time for renal adaptation to occur.* The blood pH, therefore, is the measurement that identifies the nature of the disturbance. The blood CO_2 level then defines the cause. Together these two values permit the physician to institute physiologic therapy.

* It should also be noted that renal adaptation for chronic respiratory dysfunction can at times be almost complete. In these instances, the pH will be normal but a high or low CO_2 will indicate that long standing hypo- or hyperventilation exists. Conversely, respiratory adaptation for a metabolic imbalance is seldom complete and even in slowly developing metabolic acidosis or alkalosis, the pH will indicate the nature of the disorder.

It has been maintained that adequate orientation can be obtained by a careful appraisal of both the patient's clinical problem and an accurate blood CO_2, CO_2 combining power, or serum bicarbonate, and that direct measurement of the blood pH is "rarely necessary" so long as one remembers that both metabolic and respiratory disorders can alter the CO_2 [2]. In uncomplicated disorders, careful clinical evaluation will permit an estimate of whether acidosis or alkalosis exists, provided that the level of blood CO_2 is altered, and may suggest the cause. Unfortunately, this is at best an estimate, and when pH determinations are obtained routinely a frequent serious disparity soon appears between many carefully reasoned clinical impressions and correct diagnoses based on the direct measurement of the blood pH [*10*]. In our experience, the value of arterial blood pH measurement is no longer a matter of theory but one of practical fact. Efforts have been made to obtain the same information by technics for measuring the CO_2 of expired air. This information may sometimes be useful but it also does not reflect hydrogen ion concentration and consequently is subject to the same errors of interpretation as is the level of blood CO_2. Proof of this is established in the following cases.

CASE REPORTS

Case I. M. S., a forty-two year old woman, underwent a cervical cordotomy for an intractable pain syndrome at 1:30 P.M. Although she was known to have had rheumatic valvular heart disease and bronchiectasis, there was no objective preoperative evidence of cardiac or respiratory insufficiency. She was prepared with Demerol® 50 mg. and atropine $\frac{1}{100}$ gr. Anesthesia consisted of Pentothol® 200 mg., N_2O and O_2, and Anectine® 40 mg. The total anesthetic time was one hour and forty-five minutes, ending at 3:15 P.M. The patient did not awaken from her anesthetic, and was seriously hypotensive. Oxygen and vasopressor agents were administered and she was returned to her room at 6 P.M. comatose, areflexic and in shock. Her blood pressure was 80/30 mm. Hg and pulse 132. It was believed that she had an injury to the spinal cord. Arterial blood was drawn at 7:30 P.M. The CO_2 content was 27 mEq./L. The pH was 7.04. She was treated with tracheostomy, intermittent positive pressure breathing with a Bennett valve using a 60 per cent–40 per cent helium-oxygen mixture [*8,11*], and she was given 7.5 gm. $NaHCO_3$ intravenously. At 11:00 P.M., the patient was awake and oriented. Neurologic examination was normal and her blood pressure was 126/60 mm. Hg, without pressor agents. Her arterial blood pH at 11:30 P.M. was 7.35.

This patient had a severe respiratory acidosis. The diagnosis was quickly established by the pH measurement. All other differential diagnoses of coma could be confidently discarded and energetic treatment instituted. Our experience indicates that respiratory acidosis is a frequent cause of postoperative coma and shock. With an arterial blood pH measurement, the diagnosis can be made quickly.

In retrospect, an examination of the blood pressure record during anesthesia disclosed a preoperative pressure of 140/80 mm. Hg. At 1:30 P.M. it was 150/100 mm. Hg, and at 2:10 P.M. there was a sudden rise to 200/120 mm. Hg. At 2:35 P.M. there was a sudden drop in pressure to 115/70 mm. Hg, and at 2:40 P.M. it was 100/60 mm. Hg. At this point, Vasoxyl® was given. The blood pressure rose to 120/80 mm. Hg. It is likely that the rise in blood pressure to 200/120 mm. Hg represented early evidence of hypercapnia and the subsequent sudden drop heralded the onset of severe acidosis. Anesthesiologists should be aware of the possible significance of such sudden changes in blood pressure.

Case II. O. R., a seventy-nine year old woman, underwent surgery for obstruction of the small bowel due to adhesive bands. No other preoperative abnormalities were noted. Preoperative laboratory studies revealed a hemaglobin of 15 gm. per cent, and a leukocyte count of 1 cu. mm. The urine contained albumin 1 plus, no sugar and the specific gravity was 1.020. The blood urea nitrogen was 80 mg. per cent. The serum potassium was 5 mEq./L. and the serum CO_2 was 29 mEq./L.

Postoperatively the patient had an uneventful course until the eighth day, when a wound abscess was discovered and drained. In the next twenty-four hours, the patient gradually became hypotensive, lethargic and eventually comatose. Laboratory work obtained October 16 revealed that the blood urea nitrogen was 50 mg. per cent. The serum potassium was 6.3 mEq./L., the serum CO_2 was 20 mEq./L. and the arterial blood pH was 7.15. A roentgenogram of the chest revealed bronchopneumonia in the right lung. Her blood sugar was 800 mg. per cent.

In this mixed metabolic and respiratory acidosis the blood CO_2 provided very little indication of the profound depression of the pH. This woman required emergency therapy for both diabetic acidosis and respiratory insufficiency. Without the pH measurement, it is unlikely that a correct diagnosis could be made even though one might suspect that diabetes plus some complication was responsible for her coma.

CASE III. J. S., a fifty-four year old man, was subjected to gastric resection. Postoperatively, signs of peritonitis developed. Peritoneal aspiration revealed free bile and a second operation disclosed rupture of the duodenal stump. Drains were placed in the duodenum and gallbladder. Following the second operation, the patient responded well initially but then became dyspneic, disoriented and hypotensive. The blood pressure was 90/60 mm. Hg. A portable roentgenogram of the chest revealed pneumonia or atelectasis of the left lower lobe. Blood chemistries on the fourth postoperative day were: blood urea nitrogen, 30 mg. per cent; serum sodium, 155 mEq./L.; potassium, 6.0 mEq./L.; calcium, 4.5 mEq./L. and serum CO_2, 16.4 mEq./L. Although it was believed that fluid replacement had been adequate, a diagnosis of metabolic acidosis due to excessive loss of duodenal fluid and bile was entertained. However, the arterial blood pH was 7.58 which identified this patient's imbalance as a respiratory alkalosis.

The frequency of respiratory alkalosis has been surprising to us. This is an exceedingly difficult condition to treat [12]. However, by establishing the correct diagnosis with arterial blood pH measurements, we have avoided aggravating these cases further by the administration of bicarbonate in an effort to treat the presumed metabolic acidosis as suggested by the low serum CO_2.

CASE IV. J. T., a thirty year old man in previously good health, was operated upon for a gangrenous perforated appendix. On the second postoperative day, mental confusion, hallucinations and Jacksonian convulsions developed with pathologic reflexes on the left side. He was also dyspneic and a portable roentgenogram of the chest demonstrated an extensive bilateral pneumonia. On that day, his serum CO_2 was normal (24 mEq./L.) but his arterial blood pH was 7.11.

A tracheostomy was performed, pressure breathing with a helium-oxygen mixture was administered and he received 15 gms. of sodium bicarbonate intravenously. These measures raised his pH to 7.27. During the next seven days, it was necessary to use continuous intermittent positive pressure breathing through the tracheostomy tube, and he received 35 to 64 gms. of $NaHCO_3$ intravenously every twenty-four hours for three days. During this time, he remained semi-comatose and delirious. His serum CO_2 gradually rose to over 45 mEq./L. However, serial pH determinations were consistently below 7.30 until, with continuous use of massive antibiotic therapy, intravenous expectorants and frequent tracheal aspiration, his pneumonia gradually cleared. His pH then returned to normal, his sensorium cleared and the patient recovered.

This case illustrates several important facts:

1. Profound respiratory acidosis is frequently manifest by neurologic syndromes including confusion, hallucinations, convulsions and coma. Both bilateral and lateralized abnormal neurologic signs have been observed.

2. The respiratory acidosis in this case could have been detected only by the pH measurement.

3. Without a diagnosis of acidosis, this patient's coma would have been attributed to "toxemia" and hypoxia. Oxygen would have been administered by tent, mask or cannula and he almost certainly would have died.

4. The serial pH measurements were of invaluable aid in this patient's management as well as in the initial diagnosis. With the administration of such large quantities of bicarbonate, the rising serum CO_2 had very little significance and only the pH indicated that the acidosis was still uncontrolled requiring continued efforts to improve ventilation and additional administration of bicarbonate. This man's case could not have been managed accurately without the arterial blood pH measurements.

5. It is the disturbed hydrogen ion concentration and not the absolute level of CO_2 in the blood that is responsible for the coma of respiratory acidosis [11].

6. This case also dramatically illustrates that a respiratory acidosis can be corrected only when adequate alveolar ventilation is restored.

CASE V. A. F., a seventy year old man with senile emphysema, was operated upon for relief of obstruction of the small bowel due to adhesive bands. He had been vomiting and nasogastric suction was started postoperatively. The following day, he was noted to be breathing poorly and became hypotensive. Because of a serum CO_2 value of 37 mEq./L., it was suspected that this patient had respiratory acidosis. An arterial blood pH of 7.60, however, identified this as a metabolic alkalosis, which was treated.

This patient's problem presents a common clinical dilemma. He had two reasons for potential acid-base disturbance, namely, respiratory insufficiency and loss of gastric secretions. Since both of these states can elevate the blood CO_2, differentiation could only be accomplished by measurement of the pH.

EQUIPMENT AND TECHNIC

Our pH determinations have been made with a Cambridge glass electrode pH meter. This is a reliable instrument with an accuracy of ± 0.01 pH units. This pH meter is attractive to us because of its stability and simplicity of operation. More sensitive instruments (± 0.001 units) are available but this degree of accuracy is not necessary in clinical

situations. Less accurate instruments (±0.05 pH units) are in frequent use. These are unacceptable since variations of this magnitude will obscure significant derangements of hydrogen ion equilibrium.

It is essential that only arterial blood be used for pH measurements [*11*]. In seriously ill patients, we have observed such large variations between the pH of simultaneously drawn venous and arterial blood specimens that we believe the pH of venous blood is completely unreliable for use in clinical medicine. With practice, blood can be obtained from the brachial or femoral artery just as readily as venous blood in the majority of patients. In some, it is more readily obtained.

The blood must be drawn anaerobically in a Luer-lock syringe to which a small amount of heparin has been added. If the pH measurement is not made immediately the blood should be refrigerated. The actual determination can be made at room temperature and corrected to body temperature. Only insignificant differences are observed by the additional (and complicating) use of a constant temperature water bath [*13*].

SUMMARY AND CONCLUSIONS

Our experience with a glass electrode pH meter for direct measurement of arterial blood pH has demonstrated to us that pH determinations are essential to the proper evaluation and management of an important number of patients for several reasons:

1. It has become apparent that serious errors of diagnosis occur if sole reliance is placed in changes of blood CO_2 in the appraisal of acid-base disturbances.

2. Potentially lethal derangements of hydrogen ion concentration can at times only be detected by direct measurement of the arterial blood pH.

3. Evaluation of variations of blood CO_2 in relationship to blood pH permits accurate identification of acid-base disorders.

4. Relatively simple, reliable technics are available for direct measurement of the arterial blood pH.

5. The pH determination eliminates the guesswork in the management of an acid-base imbalance, definitely identifies the disorder and permits the physician to apply proper therapy.

6. Finally, with awareness of the fact that hydrogen ion disturbance can produce coma or shock, the arterial blood pH measurement is valuable, even when normal, since this possibility can then be discarded in the differential diagnosis.

Acknowledgment: We wish to express our appreciation to Hurley L. Motley, PH.D., M.D. for his advice and counsel during these studies and for making available some of the laboratory data contained in this report.

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DISCUSSION

William H. Snyder, Jr. (Los Angeles, Calif.): It takes foresight, courage, persistence and intelligence to assume a role of leadership in the application of a basic principle in biochemistry to better surgical management.

The illustrative case histories which have been cited are convincing proof that utilization of the arterial pH in determining treatment in the critically ill semicomatose postoperative patient is

important. It is fair, I think, to say that it is vital. Having completed a long grueling operative procedure it is difficult for the surgeon to remember any new remedy for shock and coma when he is called to see the patient at 11 P.M.

The core of this paper gives the clue and backs it up not only with a theoretically sound argument but also with some convincing clinical proof. Let me resort to a formula:

$$\text{A (arterial pH)} = \frac{\text{B (bicarbonate of serum)}}{\text{C (carbonic acid of serum)}}$$

This is the basic equation which should be remembered at that late hour for the surgeon must be equipped to give the basic care.

The arterial blood pH is low when the patient is acidotic from any cause and high when alkalotic from any cause. It is highly significant if below 7.3 or above 7.5. There may be other explanations for the patient's plight, but if the pH report indicates acidosis or alkalosis of this degree it should be acted upon for this action is very likely to be the determining factor in the patient's prognosis.

The serum bicarbonate or CO_2 combining power or just CO_2 is familiar enough. When it is high we commonly say the patient is alkalotic and when low, acidotic. The formula indicates the fallacy of this thinking for every case. If only B is known, it is impossible to know A without knowing C. There is no satisfactory clinical laboratory determination of C. It depends on the respirations which are unreliable. Therefore, A and B must be known in order to calculate C.

The formula may be hazy in the surgeon's mind, and the calculations too difficult in the middle of the night. Therefore, the import of this paper for lasting memory can be stated simply. When the pH is critically low, the patient is in acidosis from some cause which will soon result in death unless it is corrected. Intravenous sodium bicarbonate is usually indicated and attention should be directed to the respiratory system. When the arterial pH is critically high, the patient is in alkalosis and no sodium bicarbonate or lactate is indicated. Surgeons are familiar with the high CO_2 in alkalosis from vomiting and will treat it well. When due to respiratory trouble, the treatment is problematic.

This is obvious oversimplification and is wrong, without some qualifications, but it is a guide. Remembering it will help the critically ill postoperative patient in the middle of the night.

JOSEPH F. BOYLE (closing): I would like to thank Dr. Synder for his comments. When we were in the process of formulating a manual for the resident staff at the County Hospital in Los Angeles on precisely this subject, his letter to Dr. Berne commenting on our initial rough drafts provided us with the necessary information to complete that paper.

I want to express my sincere thanks for the opportunity to have come here today to address your Association.

The Physiologic Basis of Cardiac Arrest During Anesthesia

R. W. PORTER, M.D., PH.D. AND J. D. FRENCH, M.D., *Los Angeles, California*

From the Surgical Service, Veterans Administration Hospital, Long Beach, California; and the Departments of Anatomy and Surgery, University of California School of Medicine, Los Angeles, California. This study was supported by a grant from the National Institute of Mental Health (M-2482).

ON occasion, it is useful to review for specialists skilled in one discipline the results of experiments employing the tools of quite another branch of scientific endeavor when these experiments bear on a topic of common dedication. For this reason, the data relating to the problem of cardiac arrest during anesthesia emanating from the neurophysiology laboratory are presented in the anticipation that they will be of interest to a forum of surgeons.

During the past decade, the core of grey and white matter, occupying the central portion of the brain stem and lying mesial to the long tracts which conduct primary sensorimotor information to and from the brain, has been found to be essential to consciousness [3], for destruction of this neural mass, called the reticular formation (hereafter designated R. F.), results in coma [4]. It can be demonstrated easily that this neural structure is defunctionalized reversibly and selectively by soporific drugs, since the administration of such agents does not interfere with impulses recorded in the laterally situated primary tracts while it obliterates completely responses travelling in the R. F. [5]. Thus, agents administered to a patient to render him stuperous and insensitive to surgical pain have an affinity for the R. F. and induce the anesthetic state by exerting a specific depressing action upon it.

In acting upon the central brain stem core, however, anesthetic agents modify other functions subserved by this region, as it has many in addition to maintaining the conscious, alert state. The nuclear masses and connections which serve to mediate visceral reflex activity, for example, are located within the R. F. and form an integral part of it. The evidence is clear now which indicates that the R. F. serves to mediate, control and quantify these visceral reflexes and to keep them in proper balance for maintaining the organism in its environment. This balance is disturbed, therefore, when anesthetics are administered and the anesthesiologist records innumerable changes in reflex activity as a patient passes through the various stages of induced depression.

Changes in cardiac reflex activity are particularly characteristic of drug-induced depression of the R. F. The somatic stimulus of a premature surgical incision, for example, may produce a profound alteration in the heart beat. Cardiac function, however, is even more sensitive to stimuli of visceral origin, particularly those applied to organs innervated by the vagus nerve. It is small wonder then that alterations in pulse ensue upon such vagal stimulation as that induced in the inadequately anesthetized patient by trachial intubation, cardiac manipulation or mesenteric traction. The present investigation was designed to study this phenomenon by attempting an assessment of cardiac reflex excitability as affected by the induction and abatement of deep anesthesia.

EXPERIMENTAL PROCEDURE

Experiments were performed on nineteen cats and five monkeys anesthetized with either sodium Pentothal® or ether. One vagus nerve was isolated and divided, and stimulating electrodes placed on its proximal end. The other vagus and the sympathetic nerves were left intact. Changes in the electrocardiogram resulted from electrical stimulation (repetitive negative pulses; 1 to 3 volts, 0.1 msec. duration, 50 to 100 pulses per second; train duration

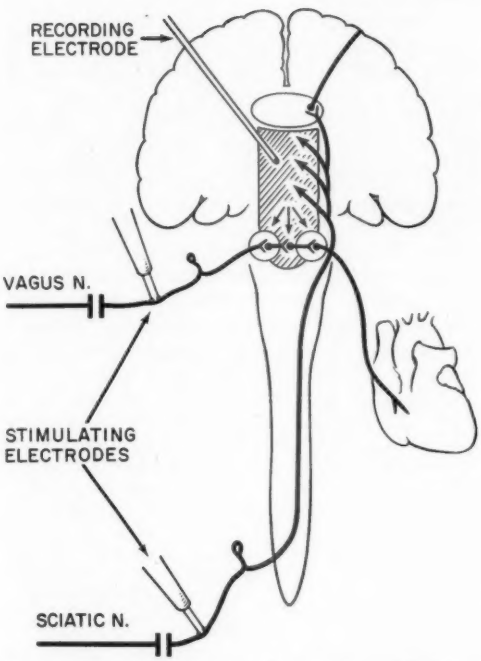

FIG. 1. Diagram indicating the experimental design used in these studies. Reticular formation (cross hatched) excitability under various stages of anesthesia was determined by recording its electrical response to sciatic nerve stimulation. This was correlated with status of visceral reflexes as determined by cardiac response to stimulation of proximal end of cut vagus nerve.

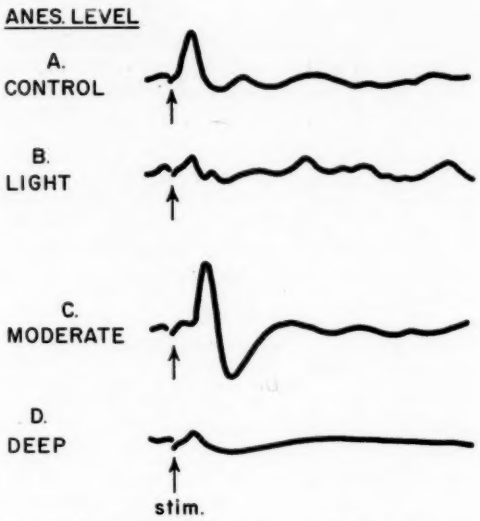

FIG. 2. Diagrammatic representation of reticular formation response to sciatic nerve stimulation under various levels of anesthesia. Cardiac irregularities could be induced by vagal stimulation only during (C) when long latency evoked response was fully manifest.

of 5 to 10 seconds) of this nerve at various levels of anesthesia. The status of this and other visceral reflexes was correlated with the functional integrity of the brain stem R. F. The excitability of the R. F. was determined by measuring the characteristics of the electrical potentials evoked within it from stimulation of the sciatic nerve (single negative threshold pulse; 2 to 3 volts; 0.1 msec. duration). (Fig. 1.)

RESULTS

Confirming previous studies, it was found that R. F. excitability, as determined by potentials recorded from an electrode placed within it, was strongly influenced by anesthesia [5]. During the control period, the evoked responses exhibited high amplitude and occurred about 20 msec. after the stimulus was applied to the sciatic nerve. (Fig. 2A.) This response was very sensitive to anesthesia and it disappeared soon after the administration of the agent was begun. (Fig. 2B.) When anesthesia was slowly induced, or given in divided doses in the case of barbiturates, a second evoked response was often encountered which was never seen in the waking state. This response was seen at a moderate depth of anesthesia and occurred some 40 to 80 msec. after stimulation of the sciatic nerve. (Fig. 2C.) This longer latency response eventually disappeared with very deep anesthesia (Fig. 2D) but potentials could still be recorded from the more laterally situated long sensory tracts from stimulation of the sciatic nerve. As the anesthetic state became dissipated the responses reappeared in the reverse order, that of long latency first and of short latency upon resumption of consciousness [2].

The cardiac rate was relatively little influenced by vagal stimulation, aside from modest slowing, during all stages of anesthesia except one. The long latency response, upon reaching maximal amplitude in the R. F., however, signaled a degree of reticular hyperexcitability which presaged the development of cardiac irregularity including complete standstill in response to stimulation of the vagus nerve. The decline and disappearance of this

same long latency potential, either as the result of the administration of more anesthetic or of the withdrawal of the agent, was attended with the reappearance of cardiac reflex stability. These phases of R. F. excitability, characterized by transient sequential appearance of the short latency and long latency responses, were easiest to recognize when barbiturates were utilized due to the greater ease with which the different stages of anesthesia could be slowly developed and sustained.

COMMENTS

In these animal experiments, cardiac arrest clearly was the result of a striking increase in the excitability of the cardiac reflex which occurred at a specific depth of anesthesia. This reflex hyperexcitability in turn correlated precisely with a change in reactivity of the R. F., signaled by the appearance within it of a long latency evoked response. It may be presumed, therefore, that the reflex hyperexcitability leading to cardiac arrest was central in origin and indicated a drug-induced distortion of visceral reflex control normally mediated by the R. F.

If these experiments can be extrapolated to man, it should be concluded that, at a stage of anesthesia, defined by the appearance of the long latency response in the R. F., the patient should be vigorously protected from all sensory stimulation particularly that applied to the vagal system. Since the surgeon and anesthesiologist will not be able to monitor an electrode in the R. F. of patients at operation, however, it will be necessary to detect the stage of reticular hyperexcitability by some other means not yet available. In animals, the period of hazard from cardiac arrest occurred when the pupil was constricted, the light reflex was still present and the carotid sinus reflex was enhanced. Hopefully it will be possible to control such visceral hyperexcitability by the administration of drugs which might depress reticular activity directly or its motor output through peripheral autonomic pathways. The matter appears to be susceptible to further clinical and experimental definition, however, and such investigations should be pursued vigorously in the interests of precision and safety.

Clinical cardiac arrest has been ascribed to a myriad of causes and there can be no doubt that many exist. However, it is very likely that the basic mechanisms involved, at least in a great many cases, are: (1) stimuli applied during (2) a stage of distorted reticular formation. Thus, all conditions which can distort R. F. function appropriately, either singly or by summation, will predispose a subject to cardiovisceral instability. Hypoxia, for example, hypoglycemia or hypercapnia, when tested neurophysiologically, all produce depression of the R. F. in a manner similar to that resulting from the administration of anesthesia [1]. These factors, therefore, summating with the many drugs administered before and during operation, may render the R. F. unusually vulnerable and the assessment of its excitability tremendously elusive.

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DISCUSSION

Howard A. Brown (San Francisco, Calif.): The authors are to be congratulated for their very stimulating and thought-provoking presentation of a subject which rests heavily upon the shoulders of every surgeon.

I should like to tell you briefly of Dr. French's background. He is qualified as a general surgeon as well as a neurologic surgeon, and while he has devoted much time to clinical surgery, his deep devotion has been to the research laboratory from which he has made many valuable contributions to the field of neurophysiology.

With Dr. H. W. Magoun of U.C.L.A. Medical School, Dr. French has organized a vast neurologic research laboratory at the Long Beach Veterans Hospital, encompassing more than 100 students working on various phases of neurologic problems. This is the largest laboratory of this type in the world.

Presently, he is professor of anatomy and clinical professor of surgery at U.C.L.A. Medical School. He is also director of the new Brain Research Institute which is being organized at U.C.L.A.

I do not know how many of you are members of the "Cardiac Arrest Club" but I assure those who do not belong that it is a most distressing and frightening experience.

I had the misfortune to have two patients with arrests in the past thirty years, both occurring within a period of ten days in the x-ray department while spinal air studies were being made under sodium Pentothal anesthesia. Cardiac massage was undertaken in both, one patient recovering completely, the other surviving for twenty-four hours. Fortunately, the latter was a patient with severe mental deterioration who had a hopeless prognosis.

Drs. Porter and French have pointed out new and interesting physiologic observations in the reticular formation in various stages of anesthesia, indicating that minimal stimuli of various types may precipitate cardiac arrest. It becomes even more apparent then that the surgical procedure itself is not the major consideration but the condition of the patient with reference to his anesthesia and the physiologic changes that this produces are probably the critical factors in cardiac arrest.

This subject was excellently presented by our President (Dr. Berne) and his associates at this meeting in 1955. Most of us were amazed at the apparent increasing frequency of this condition. Dr. Berne reported one cardiac arrest in every 585 surgical procedures at the Los Angeles County Hospital and a recovery rate of only seventeen cases of fifty-seven in which resuscitation was attempted. He also pointed out the physiologic changes occurring under anesthesia, urged that greater attention be given to the patient's preoperative condition and stressed the need for careful attention to induction of anesthesia, adequate airways and oxygen exchange, and drug dosage and other possible contributing factors.

The authors' concepts undoubtedly will add materially to a better understanding of this critical problem in the future. I am hopeful that further study, both from the standpoint of anesthesia and the neurophysiologic impact it has on the nervous system, will eventually give us a more definitive answer to the etiologic factors involved, and thereby lighten the load on the shoulders of all surgeons.

WILLIAM P. MIKKELSEN (Los Angeles, Calif.): In the paper presented here five years ago by Dr. Berne and me, we believed that little evidence existed to indict vagal effect in the genesis of cardiac arrest. The present paper naturally has stimulated our interest.

The question I wish to ask is whether the vagal stimulation used in these experiments was prolonged or short. If the stimulus was but a short burst, did the induced cardiac arrest persist after discontinuation of the vagal stimulus? If this latter situation was obtained, the authors' presentation is of great significance. If it does not, the significance is open to question.

J. D. FRENCH (closing): To answer Dr. Mikkelsen, a train of stimuli were applied to the vagus nerve, but a short burst rather than a continuing train. Sometimes when this train was terminated the heart did not start again.

Postoperative Complications

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From the Department of Surgery, Swedish Hospital, Seattle, Washington.

ANY medical center, whether it be a university or a private hospital, has the same responsibility regarding patient care, teaching and research. It is important that each know its accomplishments as well as its problems. Considering this, we propose to discuss a long-term project which has to do with the accurate recording of operative complications, the need for which is obvious both to the hospital and to its staff. The need is especially great in the hospital taking on the obligation of a prolonged surgical resident training program.

In recent months there have been those who have seen fit to comment editorially [1] and otherwise on the need for developing ways and means to reduce complications and to improve methods to prevent those which occur following the common and more frequently performed surgical operations. Several surgical departments in this country do publish annually a review of the year's work with comments on current problems. However, a review of the literature of recent years gives no picture of the over-all problem and incidence of postoperative complications. The tendency is for clinical reports more often to emphasize procedure and satisfactory results in the defense thereof, rather than acknowledge failures and accurately record complications.

One facet more recently being examined and studied most thoroughly is that of wound healing and infection.

We believed it desirable to know the present incidence of complications, particularly wound infections, as they are now occurring and are reported in The Swedish Hospital in Seattle. This paper is designed to serve as a basis for comparison in the future as a prolonged careful recording of complications is brought about. To better accomplish this we acknowledge the need for an improved method of recording such data. We are also faced with the perennial problem of the need for uniform wound description and classification. Altemeier [2] and others have continued to bring this need to our attention, particularly with reference to the staphylococcal infections.

It would seem that there might be strength in a consistent and uniform interpretation of the problem should this study be an effort limited to the authors. With the exception of certain information obtained from the wound infection committee, this has been the case. This latter information has been obtained under the direction of Drs. John B. Holyoke and John H. Koler. The majority of the data reported herein was not collected during the course of a planned study. The statistics were compiled in retrospect after a review of the surgery of 1958 and 1959. These records were checked against the thorough monthly surgical staff meeting reports and with the records of the infection control committee.

We believed that such information might best be recorded and organized using the so-called punch-card technic, devoting the information of a single card to each postoperative complication. For the present, at least, this is a more practical method than the IBM technics. In 1958 and 1959 every complication was evaluated that could possibly be gleaned from the records of this 375 bed private hospital. These records were examined in detail by the personnel of the record room and by a conscientious record committee, with further scrutiny by the house staff and residents assigned to this task. Finally, all records with any hint of a complication whatsoever were examined, after which we coded and recorded the information.

We have used a four-column code taken from the World Health Organization's "Manual of the International Statistical Classification of Disease, Injuries, and Causes of Death," using

in conjunction a Code of Operations as developed for such purposes at the State University of Iowa Hospital by Dr. Lucian E. Morris at the instigation of Dr. Nathan Womack [3].

A total of 1,416 complications have been coded in such a manner that each surgeon or his respective service may easily evaluate his or its own. Such information as length of operative procedure, type of anesthetic, blood utilization, incidence of thrombophlebitis, wound suture and multiple other facts regarding the wound and the patient are included. Using this technic, a fairly rapid and accurate evaluation of an operative procedure may be available upon short notice. As an example, it was possible to avail ourselves of the information such as recorded (Table I) fairly hastily. In another instance, for the benefit of the urologists, we were able to check quickly their record of postoperative bleeding and mortality (Table II) as it related to the several methods of prostatectomy. The individual surgeon may check his own problems. (Table III.)

In 1958 and 1959, 9,023 major and 7,855 minor procedures were performed. (Table IV.) The work with rare exception has been done by surgeons. These totals exclude procedures by the gynecologist, non-operated fracture reductions and ophthalmologic procedures. In this instance, the wound has been assessed under two main headings, namely, nature of the wound and degree of infection. The former designates whether the wound is clean, potentially infected or is frankly infected. The degree of infection is assessed as trivial, moderate or severe. Trivial refers to redness of the wound with stitch infection and even minor purulent exudation or drainage. The moderately infected wounds included all those more extensively involved with or without systemic manifestation, with or without prolonged hospital stay, and requiring treatment. Those infections grouped as severe produced a severe morbidity and were in general very extensive. Perhaps the term "major" would better include both the moderately severe and the severe infections. It is immediately evident that our surgeons are reluctant to record the presence of trivial infection. Furthermore, it is difficult to relate wound infection to total operative wounds on a percentage basis, inasmuch as we have no accurate recording of the wound status at the time of surgery. A total review of all wounds using the records in retrospect is inadequate.

It has been gratifying to find relatively few infections among our clean postoperative wounds. (Table v.) How many have occurred and not been recorded? How many have occurred and are noted only after a short hospitalization, hence, are never recorded in

TABLE I
COMPLICATIONS AND MORTALITY RESULTING FROM RANDOM OPERATIVE PROCEDURES

Procedure	No. of Cases	Complications	Mortality
Repair of inguinal hernia	738	36	1 (0.13%)
Gastric resection for peptic ulcer	224	45	7 (3.1%)
Operation for carcinoma	37	12	2 (5.4%)
Prostatectomy	899	150	14 (1.5%)
Colon resection	121	55 (50 patients)	16 (13.2%)
Abdominoperineal resection	49	23 (17 patients)	0 (0.0%)

TABLE II
COMPLICATIONS, MORTALITY AND EVALUATION OF HEMORRHAGE IN SEVERAL METHODS OF PROSTATECTOMY

Type of Prostatectomy	No. Performed	Complications	Hemorrhage			Returned to Operating Room	Mortality
			Minimal	Moderate	Severe		
Suprapubic	103	31	0	9	1	3	1
Retropubic	389	56	0	13	1	7	1
Perineal	55	12	0	0	0	0	0
Transurethral	352	51	1	14	3	10	12
Total	899	150	1	36	5	20	14 (1.5%)

TABLE III
PER CENT OF COMPLICATIONS AND WOUND INFECTIONS ATTENDANT ON THE WORK OF EIGHT SURGEONS

Surgeon	Infection (%)	Wound Separations (%)	Complications (%)
A	3.0	0.0	15.6
B	1.3	0.6	6.8
C	1.45	1.2	12.0
D	0.4	0.0	6.7
E	4.0	0.5	11.4
F	3.1	1.5	20.6
G	3.3	0.0	12.0
H	4.7	0.8	17.4

TABLE IV
OPERATIVE WOUND INFECTIONS (178) FROM 9,023 MAJOR* PROCEDURES AND 7,855 MINOR PROCEDURES

Wound	General Surgery (8,637)	Orthopedic Surgery (3,261)	Urologic Surgery (4,430)	Neurologic Surgery (550)
Clean	24	35	3	6
Contaminated	91	5	10	
Infected	3	1		
Total	118 (1.4%)	41 (1.2%)	13 (1.2%) 41†	6 (1.1%)
Trivial	28	14	3	2
Moderately severe	80	26	10	3
Severe	10	1		1
Total	118	41	13	6

* Excluding non-operated fracture reductions, cast changes, gynecologic surgery by gynecologists, and ophthalmologic and minor otolaryngologic procedures.
† Other infections throughout genitourinary tract.

TABLE V
CLEAN WOUND INFECTIONS

General Surgery	No.	Orthopedic Surgery	No.
Varicose veins, Burn scars, Skin tumors, Postphlebitic	9	Laminectomy and/or fusion	16 (46%)
Large artery	4	Other arthrodeses and fractures with or without metal fixation	9 (26%)
Abdominal hernia	3		
Breast	2	Other	10
Intestinal obstruction	3		
Clean bone	3		
Total	24	Total	35

the hospital? We will not know the answer to these questions until the planned study now in progress is completed.

We are fortunate in having available information (Table VI) carefully obtained by the physicians participating in the infection control program. During the period of this study, the organism, coagulase-positive hemolytic Staphylococcus aureus, was cultured from 529 wounds. Evaluation of the committee at the bedside determined 127 of these to be hospital-acquired. These involve both medical and surgical admissions numbering 36,000 during the same two-year period. There were seventy-nine significant postoperative wounds infected with coagulase-positive Staph. aureus. These excluded such acquired hospital infections as furuncles and those in wounds unrelated to the wound involved in the surgery. Of these, forty-three were considered clean, uncontaminated wounds. The remaining thirty-six were a part of a contaminated wound.

Phage typing during the same period revealed a pattern consistent with that in most hospital infections, namely, the great majority of antibiotic-resistant staphylococci isolated in this hospital belonged to International Phage Group III. (Table VII.) This is consistent with the universal observation wherever phage typing has been applied to studies of the incidence

TABLE VI
COAGULASE-POSITIVE STAPHYLOCOCCAL INFECTIONS

Data	No.
Total hospital admissions	36,000
Total coagulase-positive Staph. aureus wound cultures	529
Infections considered hospital-acquired	127
Significant postoperative wound infections	79
Significant clean wound infections (hernia, laminectomy, etc.)	42

TABLE VII
INTERNATIONAL GROUPING OF TYPABLE CULTURES

Type	Not Hospital-Acquired (No.)	Hospital-Acquired (No.)
Group I	7	9
Group II	4	5
Group III	45	25
Group IV		
Phage 80–81	35	18

TABLE VIII
POSTOPERATIVE WOUND INFECTION

	Year	Total	Clean	Moderate and Severe	Per cent	Trivial	Per cent	Total	Per cent
Meleney, New York (1935).....	1925	558	482	20	4.0	56	10	76	14
	1933	1,132	1,078	13	1.1	41	3.6	54	4.8
Howe, Boston (1954).....	1949		796		0.3		0.4	5	0.7
	1953		800		1.87		0.37		2.25
Clarke, England (1957).....	1954		382	25	6.5	27	7.1	52	13.6
Shidler (1959).....	1958		546					5	0.9
Swedish Hospital, Seattle (1960)...	1959	8,637	6,621	77	1.2	26	0.4	103	1.6

of staphylococcal infection. Group I is next in frequency with the familiar 80–81 phage, a part of both groups I and III.

Finally, we have compared our infection rate to other hospitals, as much as any comparison is possible. (Table VIII.) This is a comparison not unlike that of Jeffrey and Sklaroff [4]. For the purposes of this evaluation we are considering procedures such as herniorrhaphy, mastectomy and sympathectomy as clean. Operations in which there is actual or potential contamination but in which the wounds are normally expected to heal primarily are also included. These would include bowel resection, cholecystectomy, perineal surgery and skin grafts.

Meleney [5], in a planned study, evaluated the wounds of nine years' surgery beginning in 1925. In a retrospective study Howe [6] evaluated a group of patients considered clean. His definition of a clean wound is just as described. Clarke's [7] series of wounds exclude any of those drained. Shidler [8] evaluated 546 operative wounds or 25 per cent of 2,200 operative wounds created during four months of 1958 at the Palo Alto Hospital. This group of operations is not dissimilar to others included in Table VIII. For purposes of comparison, similar procedures performed during 1958 and 1959 at the Swedish Hospital are included. In view of the many variables in making such comparisons, we believe a careful planned study of infection rates is in order.

We plan to continue the present study in order that we may eventually have an accurate appraisal of operative complications, including a careful classification and an idea of the incidence of wound infections as they occur in a particular moderately large hospital.

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DISCUSSION

ALLAN D. MCKENZIE (Vancouver, B. C.): Dr. Lobb took a chance indeed when he asked me to discuss his important paper. First, as a fellow neophyte, I qualify not as a blue chip but rather as a highly speculative risk. Second, as a Canadian I move into the wildcat class, for a fellow Canadian, Dr. McKinnon of Toronto, recently won notoriety by proving that all our efforts have not improved the prognosis of cancer of the breast.

To review our failings is always most salutary. I would agree with Dr. Lobb that we are prone to preen ourselves on our accomplishments, but fail to flagellate ourselves on our failures.

There are a few points to mention about a study such as this:

1. Collection and evaluation of data must be done by ourselves if it is to be significant. Information from record room reports may be helpful, useless or frankly misleading. This entails a lot of hard

work on the part of a dedicated person such as Dr. Lobb, and I believe the Swedish Hospital is fortunate to have his enthusiasm.

2. The information studied must be limited in volume and scope. This work concerning a 375 bed hospital falls within reasonable limits and will result in valuable observations. A comparable study of our own 1,800 bed institution in which 25,000 operations are performed annually would not prove much. We might confirm Parkinson's law that work output varies adversely with the number of people employed.

3. Some problems, e.g., wound infection, must be documented while the patient is in the hospital.

4. Figures obtained must be interpreted with the mental reservation that we are dealing with complex individuals—patients and surgeons—beset by complex problems. Figures can be made to prove anything we wish to believe, an art widely practised by ardent advocates the world over.

The reported incidence of wound infection is very good. It is difficult to compare it with our own figures and I do not believe it is profitable to so do. We have used a different classification of wound infection, listing anything which falls short of ideal wound healing. There are four grades of infection: (1) reddening, (2) pustule formation, (3) frank infection of part of the wound and (4) frank infection of the whole wound usually with some systemic reaction. Again, our cases have been accumulated while the patients were in the hospital, and it has been our experience that many cases previously missed are found this way. The cases studied involved major surgery, in fact, quite extensive surgery formed a large proportion of them. We have noted that extensive head and neck surgery is fraught with an appreciably higher incidence of staphylococcal infection, an observation which has practical applications in trying to eliminate this organism preoperatively from the upper respiratory tract. In lengthy (over three-hour) abdominal operations there was an appreciable increase in wound infections due to organisms other than staphylococcus. This observation has practical applications with respect to the length of surgery and protection of the wound during surgery.

Dr. Lobb's table of mortality from various operative procedures caught my eye, in particular, the figures for colon surgery and gastrectomy for peptic ulcer. The former is high, as I suspect is our own, and I cannot help but wonder if this is not the price paid for our more radical (no colostomy) attack on colonic lesions. Our mortality rate for gastrectomy for peptic ulcer was 3.7 per cent, slightly higher than that reported by Dr. Lobb. My rationalization of this discrepancy extended only to one feature, namely, our aged population. Improbable as this may seem, British Columbia is regarded as the banana belt of Canada where all those who can, direct their footsteps in retirement. This is reflected in our population, 10.5 per cent of which are over sixty-five, in contrast to Canada as a whole where the figure is 7.6 per cent.

MAX GASPAR (Long Beach, Calif.): I, too, think Dr. Lobb is to be highly congratulated for bringing us this rather negative type of report. I know how hard it is, because we have been doing this for some years on the College of Medical Evangelists service at the Los Angeles County Hospital for the worst complication of all, namely, death.

We have studied all the mortalities since 1952 and have made an effort to classify the causes of death into broad general groups. Each patient who dies is studied very carefully, with the resident or residents involved, as soon after the death occurs as possible while all the facts are fresh in our minds.

We classify deaths as those due to faulty preoperative care, errors in diagnosis, judgment and operative technic, anesthesia, faulty postoperative care and patient's disease. If one of these was principally responsible for the patient's death, it is recorded as the primary cause of death, but if only partially responsible, it is recorded as a contributory cause. I would like to point out that errors in judgment and operative technic were alluded to by our President in his address. We believe that these have been very important for us to study. We hope we are learning lessons.

The largest group by far is that in which the patients died because of their disease process. That goes along with what was just reported from Canada. We, too, have a very aged population at the County Hospital, and naturally most of the patients die because of their own disease processes; nevertheless we obtain value from studying these cases and trying to classify them.

W. WALLACE GREENE (San Francisco, Calif.): I believe it is essential that the complications, unusual occurrences and problems in management encountered in the treatment of patients be reviewed regularly by the staff of each hospital. This data should be reviewed and analyzed by some delegated committee rather than be compiled by the record department. Any pertinent or instructive information should be channeled through this committee to the staff.